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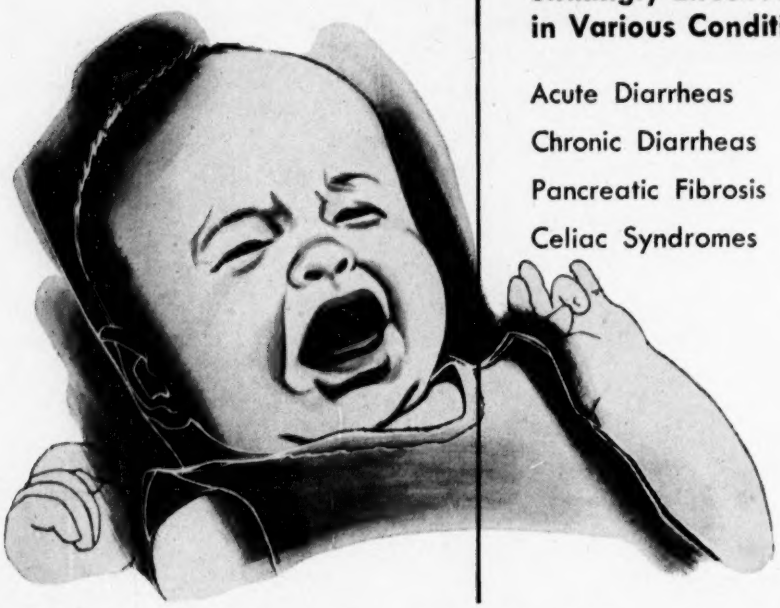

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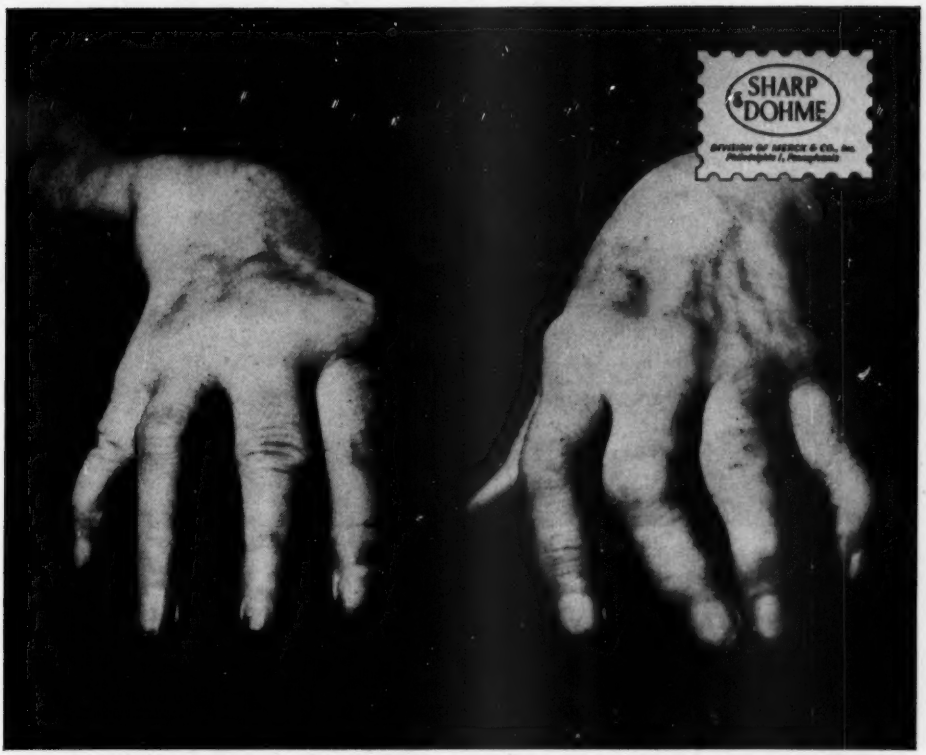

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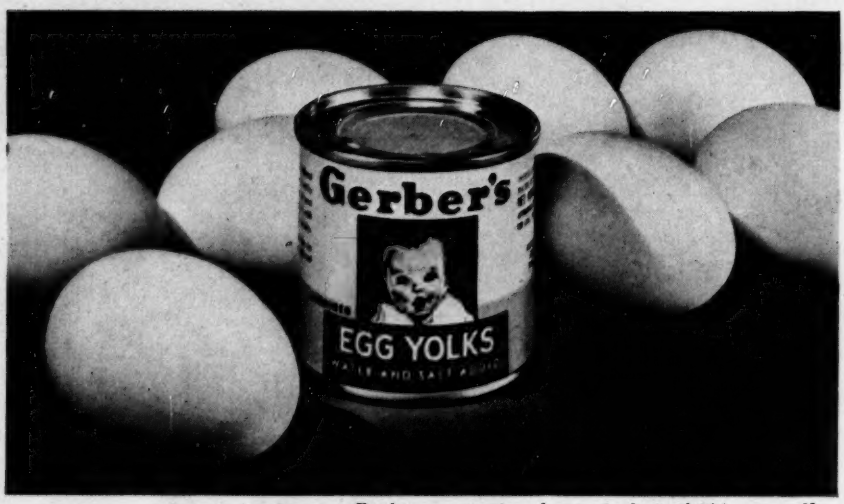

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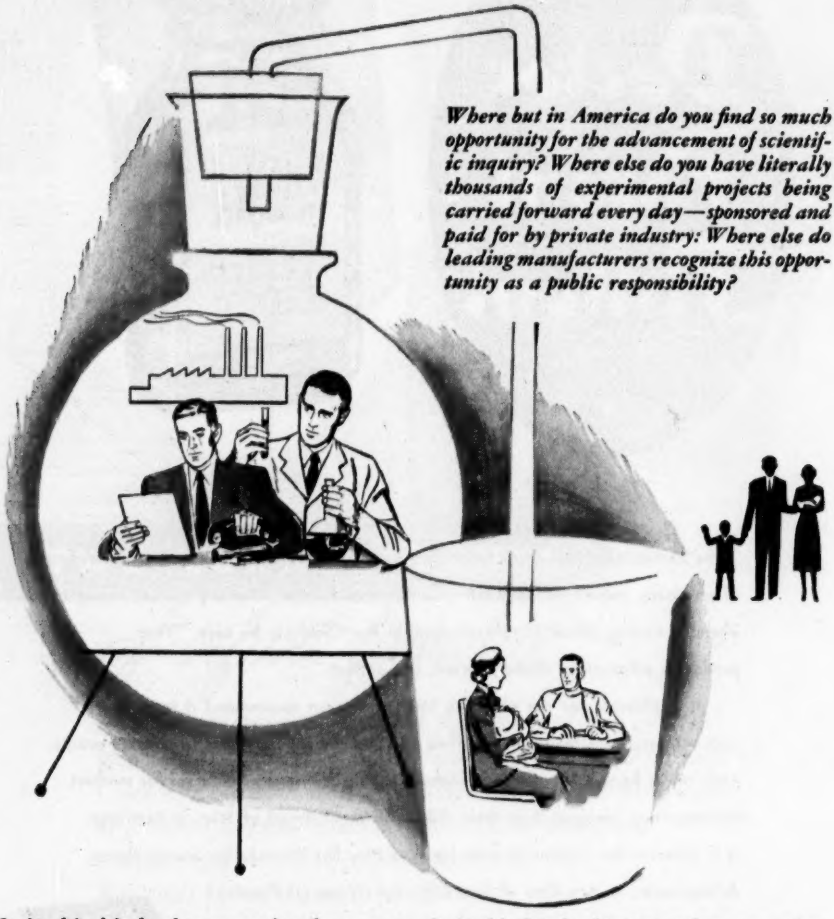

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VOLUME 87 JANUARY 1954 NUMBER 1

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FOOD POISONING IN INFANTS CAUSED BY EGG-YOLK POWDER

HAROLD ABRAMSON, M.D.
MORRIS GREENBERG, M.D.
SAMUEL PLOTKIN, B.S.
AND
CAROLYN OLDENBUSCH, M.A.
NEW YORK

DURING the last quarter of 1952, there occurred in a number of cities throughout the United States [1] cases of food poisoning in infants one year of age and under. The incriminated food was a canned egg-yolk-powder preparation, and the offending organism, Salmonella montevideo. Thirteen babies became ill with fever and acute diarrhea in New York City. The epidemiological and clinical features of these cases form the basis of this report.

On Nov. 20, 1952, the U. S. Food and Drug Administration was informed of the occurrence of salmonellosis in an infant in Washington, D. C., who had received egg yolk as part of his diet. Salmonella montevideo was recovered from the child's stool and from the egg-yolk powder. Additional cases cropped up in other cities.[1] On Nov. 28, this information was given to the office of the Assistant Commissioner for Environmental Sanitation of the department.* On Dec. 2, the Bureau of Preventable Diseases was alerted to two possible cases in infants.†

Investigation of the two infants was begun the same day. In addition, a request was made of the laboratory of the department to report the names of all persons whose stools were submitted and yielded Salmonella montevideo on culture. A similar request was addressed to the Salmonella Center at Beth Israel Hospital.‡ Inspectors of the Bureau of Food and Drugs of the department had already been assigned to retail groceries, drugstores, and hospitals to embargo any cans that might have been on shelves. They also supplied names of infants with possible cases. A few notifications were received from physicians. In all cases, definite or suspected, inquiry was made by field epidemiologists into the occurrence of acute diarrhea and other symptoms, the ingestion of canned egg-yolk powder, and the time relationship between the both. Histories were obtained from the physician, at the home of the infant, and from the hospital of admission, whenever the patients were hospitalized. Arrangements were made for the collection for culture of stools from the patients and all members of their households. All canned egg-yolk powder in the homes was confiscated and forwarded to the laboratory for culture.

From the Bureaus of Preventable Diseases, Food and Drugs, and Laboratories, New York City Department of Health.

*Federal Security Agency, Food and Drug Administration, Telephone communication of Nov. 28, 1952, from Eastern District Office in New York City.

†C. C. Dauer, Medical Advisor, Public Health Service, Federal Security Agency, personal communication.

‡Dr. Ivan Saphra, of the Salmonella Center, assisted with this study.

CLINICAL CONSIDERATIONS

As a result of these investigations, there were found in the city 13 cases of acute diarrhea in infants who had received an egg-yolk-powder preparation prior to onset of illness. The dried egg yolk was stirred into warmed feedings. All babies had been previously in good health, with the exception of one who had had a celiac type of diarrhea since the age of 5 weeks. The diarrhea in this instance was acutely aggravated.

Age, Sex, and Color.—One case was in an infant 4 months of age, six in infants 6 months of age, and the remaining in infants from 7 to 12 months of age. Ten of the babies were girls, and all but one were white.

Time Intervals.—The interval between first ingestion of egg yolk and onset of diarrhea could be estimated in only eight instances and varied from the day of initial ingestion of the powder to 29 days later. The other five babies had indefinite histories of having received egg-yolk powder for a period of from one to five months prior to onset of acute illness. Three babies had onsets in October, seven in November, and three in December.

Clinical Course.—The onset of illness was acute in all babies. Fever was prominent, ranging in 5 cases from 104 F. (40 C.) to 105.6 F. (40.9 C.). In six other babies fever varied from 100 F. (37.8 C.) to 103 F. (39.4 C.). One infant had a low-grade fever, and another had no known temperature rise. All infants had diarrhea. The stools were frequent, loose to watery, and bright yellow to green, with occasional presence of mucus. Blood was seen in the stools of only one infant. Vomiting was noted only once. Other symptoms consisted of occasional irritability, anorexia, colic, and rhinitis. The duration of illness could not be timed accurately because of difficulties in determining when the diarrhea subsided. The time of recovery was variable and was said to range from 3 to 83 days. Eleven of the infants were well within three weeks. Of the 13 infants, 5 were admitted to hospitals, where they remained from 1 to 18 days.

Blood cell counts were done for four infants. In three they were essentially normal; in one there was a leucocytosis of about 19,000, with 56% lymphocytes. Salmonella montevideo was initially recovered from the stools of 10 babies from 1 to 52 days after onset of diarrhea. In only one instance was a culture of Salmonella montevideo recovered from a household member. This was in a father who was asymptomatic. Five samples of egg-yolk powder were obtained from the homes of the babies. Only one sample grew the Salmonella montevideo on culture. The baby in this family also had a positive stool culture.

Recognition of the 13 cases was therefore based on recent ingestion of egg-yolk powder in feedings, sudden onset of illness with fever and acute diarrhea, and the recovery of Salmonella montevideo from the stools of 10 babies. Of the three babies with negative stool cultures, two also had negative egg-yolk-sample cultures. No egg-yolk sample was available in the third baby. These cases were included because of the history of recent ingestion of egg-yolk powder and the occurrence of subsequent acute diarrhea. Treatment consisted of controlled feedings, maintenance of fluid and electrolyte balance, and the use of sulfonamides and antibiotics. There were no deaths.

As a parallel procedure, there were investigated eight children from 1½ to 13 years of age with acute diarrhea from whose stools the laboratory had recovered

one of the following Salmonella organisms: S. typhimurium, S. cholera-suis, S. oranienberg, S. enteritides, and S. montevideo. None of the children was found to have had egg-yolk powder.

EPIDEMIOLOGY

Manufacture of the incriminated egg-yolk powder was started in the United States in May, 1952.[1] The product was first distributed in July, 1952, almost entirely through wholesale dealers to grocery stores and possibly hospitals in larger cities of the United States. It was marketed in sealed cans of 1½ oz. (45 gm.) net weight. Each can contained approximately 18 teaspoonfuls (75 ml.) of dried egg yolk, equal to four and a half or more egg yolks. Instructions on the label directed the thorough mixture of 2 to 4 teaspoons (8 to 16 ml.) in a serving of ready-cooked or cooked cereal, strained vegetable, strained meat, or soup. Instructions were also printed for use of the egg-yolk powder in recipes. For each fresh egg yolk required in a recipe, a mixture of 4 teaspoons of dried egg yolk and 1 tablespoon (15 ml.) of water was recommended.

Initial reports of illness were made to the Public Health Service in November, 1952. As of the week ended Jan. 24, 1953, a total of 83 cases were reported from 17 states and the District of Columbia. In 45 of the infants there was laboratory confirmation of infection with Salmonella montevideo, while in 38 infants the diagnosis was made clinically only. Bacteriological culture was made of samples of various lots of the entire production of egg-yolk powder. Salmonella montevideo was recovered from many of the samples, but contamination was not uniform in all lots. Manufacture of the egg-yolk powder was discontinued. Recall of the product was completed in December.[1]

A total of approximately 72,000 cans was embargoed in New York City. About 300 unopened cans were forwarded for culture to the laboratory of the department. These consisted of three to four samples from each of 85 lots embargoed and comprised 50 different code numbers. Cultures were made of the entire contents of one can of each code number. Eleven yielded Salmonella montevideo. From one can there were cultured two organisms, S. montevideo and S. urbana. Four more cans were then selected at random from the 11 code numbers found positive and similarly cultured. From two of these S. montevideo was recovered. This would seem to indicate that contamination was not uniformly distributed in all cans bearing the same code number.

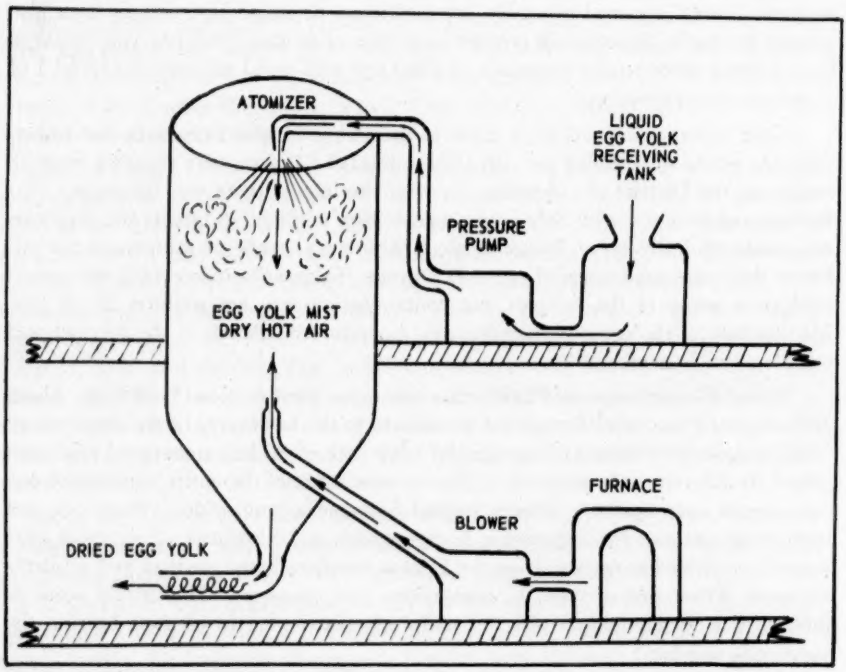
Manufacture of Dried Egg Yolk.—Prior to World War I, the manufacture of dried egg products was a growing industry in the United States.[2] During the conflict, the industry suffered. China then became the center of manufacture of egg powder. A revival of the industry in the United States occurred during World War II, with increased demand for egg powders for use here and for shipment overseas.

Drying of whole egg, egg yolk, or egg albumin is accomplished by the spray-drying method in specially designed machines.§ The initial step in manufacture is an individual hand process. Eggs are received from the farm or market and are at times cleaned. Girls inspect the eggs by candling them. The eggs are then cracked manually over a special cracking cup which is also used to separate yolk from

§ Mr. Abraham E. Abrahamson, Chief, Wholesale Division, Bureau of Food and Drugs, New York City Department of Health, assisted with technical aspects of egg drying.

albumen. Discard is made of eggs with bad odor and with defects. All equipment is washed and steamed once or twice a day, as well as after contamination with inedible eggs.[2] In this preliminary phase the handling of a single infected egg may soil the hands of the operator and thus contaminate an entire batch of eggs if adequate handwashing is not observed. The cracking of eggs may be carried out in one plant and the process of drying in another plant located elsewhere.

The whole egg or egg fraction is sometimes then pasteurized. By definition, pasteurization of milk and milk products implies heating either to 143 F. (61.7 C) for 30 minutes or 160 F. (71.1 C.) for 15 seconds, with immediate cooling to at least 50 F. (10 C.). If egg yolk or albumen is subjected to these temperatures, it is



Schematic drawing to illustrate general arrangement of one type of spray-drying apparatus for dehydrating egg yolk.

rendered insoluble and loses palatibility.[2] Experimentally, when whole egg was heated to 145 F. (62.8 C.) for one minute, all coliform organisms were killed.[3] Similar results were obtained with egg white at a temperature of 134 F. (56.7 C.) for 1.6 minutes, and for egg yolk at 142 F. (61.1 C.) for 2 minutes.[4] When practiced, pasteurization of egg yolk is carried out at 142 F. for two minutes. If these standards were rigidly applied to the manufacture of dried egg products, a safe product should be possible. However, because of the frequency of reported outbreaks from dried eggs, it is doubtful whether efficient pasteurization is uniformly achieved.

After pasteurization of the whole egg or egg fraction, the liquid is poured into the receiving tank of a dehydrator. Several drying machines of different design are

in use.[5] In general, the liquid egg is forced by a pump developing from about 2,500 to 4,000 lb. of pressure, up to the top of the dehydrator. The material is then impelled through narrow-gauge jet nozzles into the drying chamber as a mist. At the same time, dry air heated from about 185 F. (85 C.) to 200 F. (93.3 C) is blown into the bottom of the dehydrator chamber. Simultaneous dehydration of the egg is accomplished at the level of contact.‖

The egg falls in powdered form to the conical bottom of the drier, from which it is conveyed by mechanical means. The powder is left to cool at room temperature and is then packed and sealed under the usual canning precautions. Samples of the dried egg are taken for culture. The process of dehydration kills many bacteria. Others die during holding, if the moisture content is kept low. The drier the product, the less chance of multiplication of bacteria. The final moisture content of the product may not legally exceed 5%. The application of air heated to 185 F. to 200 F. to the egg droplets during dehydration should make for a safe product. However, rapid cooling occurs. The amount of heat needed for evaporation is so great that little is available for sterilization. Even the exterior of the dehydrator chamber may not become heated above 120 F. (48.9 C.).‖ Under these conditions, the dehydrator may actually become an incubator. The drying operation should therefore not be regarded as a substitute for proper preliminary pasteurization of the liquid egg, nor should pasteurization replace good raw material or good sanitary practices.[8]

COMMENTS

Outbreaks of acute gastroenteritis from the ingestion of powdered-egg products are not unusual. Attention is called to their frequency in the monograph by the British Research Council.[9] Solowey and McFarlane[10] cultured Salmonella organisms from 35% of 5,198 samples of spray-dried whole-egg powder obtained from various dehydrating plants from 1943 to 1945. Fifty-two types of Salmonella organisms were identified, with five predominating in 73% of the isolations, namely, S. pullorum, S. oranienburg, S. montevideo, S. tennessee and S. anatum. The organisms were culturally and serologically similar to those isolated from humans and from animals.

In the United States there is widespread infection of fowl with potential food-poisoning types of Salmonella.[11] Eggs and egg products are therefore frequently infected. Contamination may occur either during passage of the egg in the oviduct or by penetration by organisms through the permeable shell and membrane as the fresh warm egg cools when brought into contact with fluid containing fecal matter at lower temperatures than the egg.

Where powdered whole egg or egg fractions are employed in recipes that require baking or boiling for prolonged periods at high temperatures, reasonable safety in use may be assured. However, where dried egg products are taken from the can and stirred into warm broth, cereal, vegetables, or other food used for infant feeding, a hazard results, particularly in infants, who are very susceptible to diarrheal disorders. Until methods are devised to pasteurize or sterilize dried whole egg and egg yolk and white adequately, their use in infant feedings should be discontinued, unless facilities for appropriate terminal heating or sterilization of the feedings are available in the home.

‖ References 6 and 7.

SUMMARY

In the last quarter of 1952, New York City experienced an outbreak of food poisoning from contaminated dried egg yolk. The offending organism was Salmonella montevideo. Investigations disclosed 13 known cases in infants one year of age and under. There were no deaths. The clinical and epidemiological features of the outbreak are described, as well as the hazard involved in the use of powdered-egg products in infant feeding.

The cases in New York City occurred simultaneously with cases in other cities of the United States. The Department of Health was alerted to the existence of cases through the epidemiological intelligence division of the Public Health Service in Washington, D. C. It was therefore possible to take immediate measures to withdraw the incriminated food from the market and to institute prompt measures to prevent a widespread outbreak. The value of this centralized exchange of medical and epidemiological information is demonstrated as a worth-while measure in civil defense.

125 Worth St. (13).

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URETERAL ELECTROMYOGRAPHY IN CONGENITAL MEGALOURETER

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CONGENITAL megaloureter is a disease of infants and children. Most of these patients die before 20 years of age. The cause of the disease remains obscure. There is no conclusive evidence that infection and chronic inflammation may actually cause or predispose toward its development. It is difficult also to explain the causal factor responsible for the extreme ureteral dilation and hydronephrosis in this condition on a nonobstructive basis; yet no obstruction is demonstrable at the ureterovesical juncture or bladder neck. If obstructive uropathy is demonstrated, the ureteral dilation is usually termed secondary, and the case is not classified as primary or congenital megaloureter. To date, the most reasonable causal factor proposes a faulty neuromuscular mechanism producing ureteral atony, frequently in association with dilation of the bladder. A comparison has been made between congenital megaloureter and Hirschsprung's disease. There is, in fact, probably more than chance association of the high incidence of these two conditions coexisting in the same patient. In congenital megacolon, the rectosigmoid contains no Auerbach's-plexus ganglion cells and probably represents a maldeveloped pelvic parasympathetic system. Swenson and co-workers [1] have investigated the problem of megaloureter from this standpoint and demonstrated a decrease in the number and size of parasympathetic ganglia in the bladder of patients with this disease. It is not known whether this anatomic decrease in the number and size of parasympathetic ganglia would sufficiently alter the physiology to produce the marked vesical and ureteral atony observed in these patients. To pursue the problem further from the functional aspect, this paper reports an investigation of ureteral electromyography in a patient with congenital megaloureter.

MATERIAL AND METHODS

The patient studied presented the standard criteria for the diagnosis of congenital megaloureter. She was first examined and treated for pyelonephritis at the age of 4 years. At that time intravenous pyelograms revealed marked bilateral hydronephrosis and ureteral dilation and a large bladder. At the age of 15 the patient was first examined at the University of Chicago. Neurological examination was normal. Intravenous pyelograms failed to produce visualization of the kidneys. Cystoscopy revealed a bladder of large capacity in the absence of obstruction at the bladder neck. The ureteral orifices were large and gaping. Cystogram with 1,000 cc.

This study aided by the United States Public Health Service and an American Cancer Society Institutional Grant.

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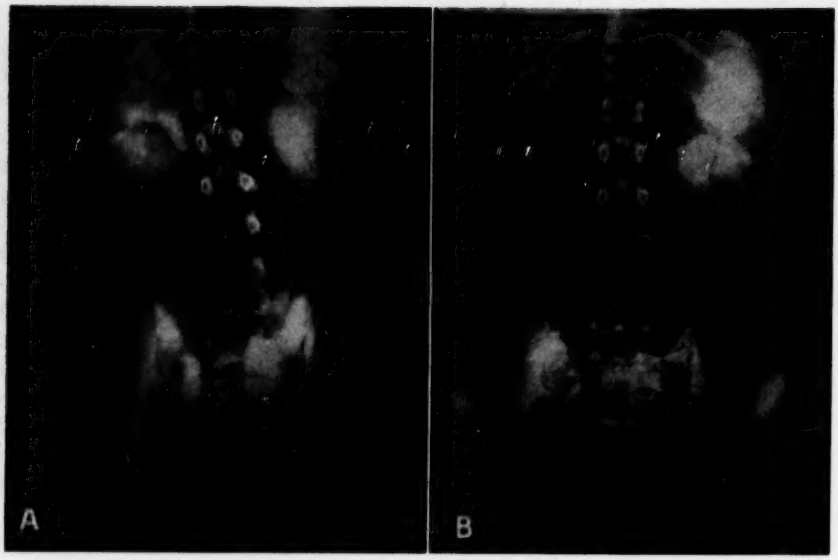

Fig. 1.—*A*, congenital megaloureter. Cystogram demonstrating ureterorenal reflux, dilated ureters, double left ureter, and marked bilateral hydronephrosis. *B*, same patient immediately after voiding. Observe decrease in size of kidneys indicating extremely thin renal parenchyma.

Fig. 2.—Electroureterogram of right ureter. Triphasic peristaltic action potentials of normal duration but probably of slightly decreased voltage.

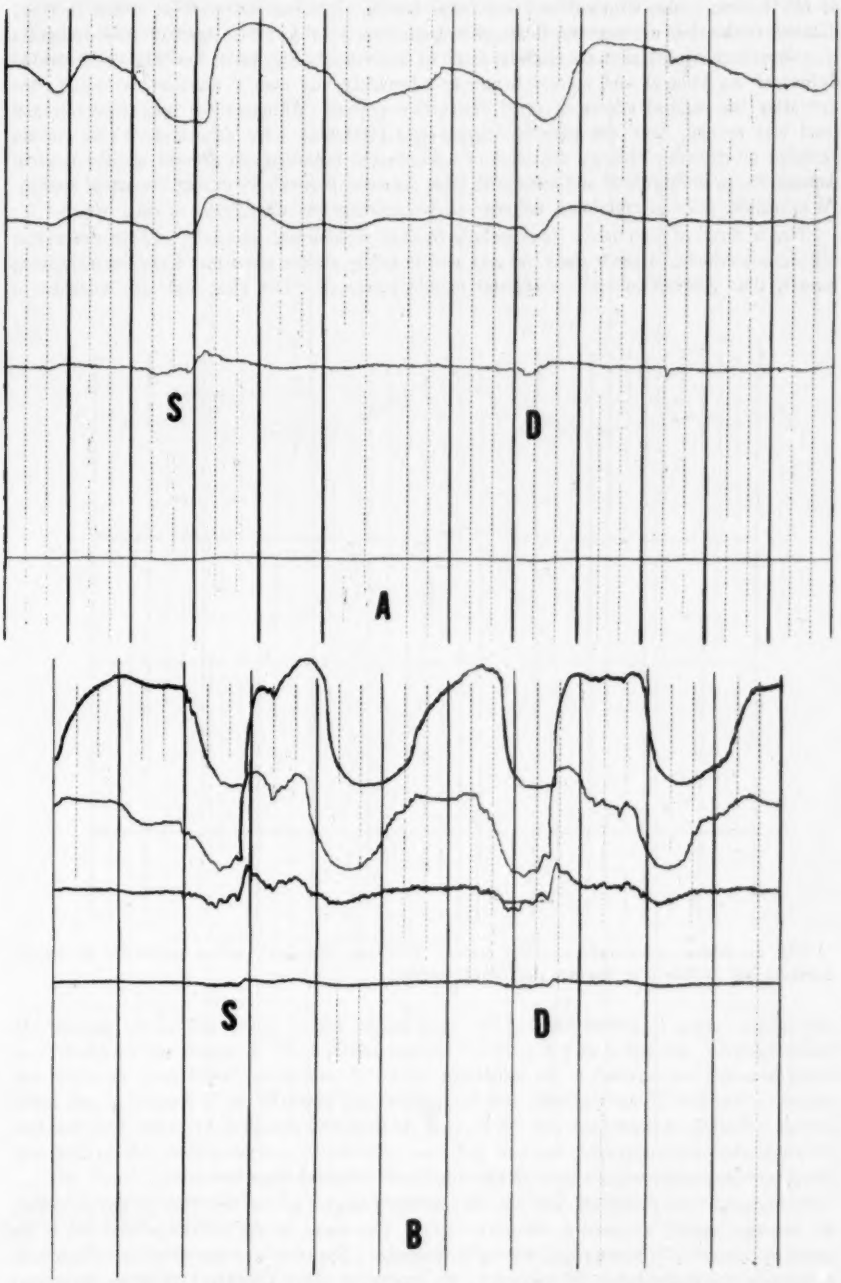

Fig. 3.—*A*, congenital megaloureter. Action potentials of occasional sustained, forceful peristalsis. Systole (*S*) and diastole (*D*). *B*, action potentials from ureter dilated as a result of ureterovesical obstruction. Observe similar prolonged, forceful peristalsis. Systole (*S*) and diastole (*D*).

of 6% sodium iodide demonstrated the large bladder, bilateral ureterorenal reflux, marked dilation, double left ureters, and bilateral hydronephrosis (Fig. 1*A*). Another roentgenogram was taken immediately after the contrast material was voided (Fig. 1*B*). This film demonstrated failure of the kidneys and ureters to empty adequately and also a decrease in kidney size indicating the marked degree of renal destruction present. Although the nonprotein-nitrogen level was normal, urea and phenolsulfonphthalein clearances were decreased. Urine culture revealed no growth. Therapy consisted of a permanent cystotomy to prevent additional renal damage due to intraureteral and intrarenal urine pressure, particularly during the act of voiding. At operation, prior to cystotomy, unipolar electroureterograms were taken of each ureter.

Two to 4 cm. of each ureter was carefully freed of peritoneum, anteriorly, and the underlying muscle, posteriorly. A small glass rod was used to gently elevate the ureter from the underlying muscle, thus eliminating artifact skeletal muscle potentials. The glass rod was attached to

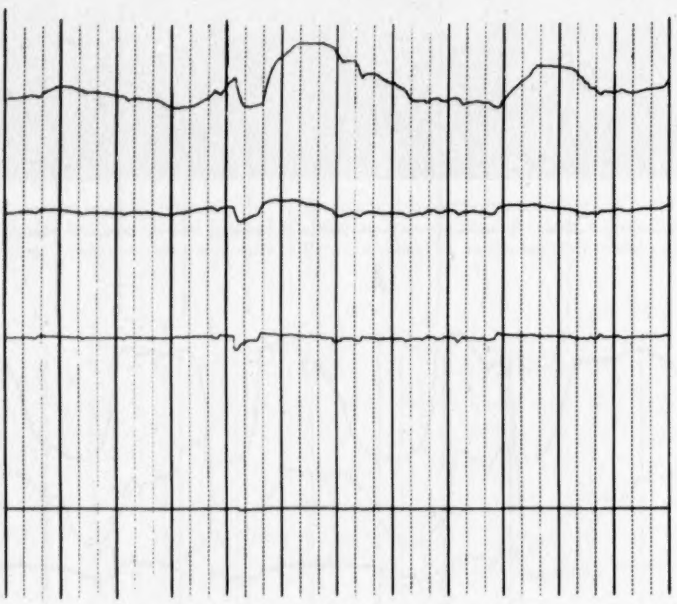

Fig. 4.—Electroureterogram of left ureter. Triphasic peristaltic action potentials of normal duration but probably of slightly decreased voltage.

soft rubber tubing suspended between two ring stands, one on either side of the patient. An active electrode, consisting of a No. 28 hypodermic needle 1 cm. in length and attached to an insulated wire, was inserted in the suspended ureter. A neutral or "indifferent" electrode was placed in the skin of the abdomen, and the patient was grounded by a contact to one ankle. Action potentials passing down the ureter were obtained and amplified by means of a standard Offner electroencephalographic machine and were recorded by a crystographic ink-writing unit. The procedure employed has been standardized and described elsewhere.[2]

Calibration.—As amplitude was recorded at right angles to the direction of the recording, the tracings usually assumed a sinusoidal curve. The shape of the electroureterogram is the result of changes in frequency and amplitude (voltage). Duration was expressed in milliseconds. Amplitude was calibrated at 100 microvolts per centimeter above (positive) or below (negative) the base line. Determination of frequency of peristalsis was constant for each experiment as the graph paper moved beneath the recording pens at a rate of 2.5 cm. per second. Each experimental tracing usually had four channels in which various filters were inserted to progressively remove

slow frequency cycles: Channel 1, 0.5 filter; Channel 2, 0.2 filter; Channel 3, 0.02 filter; and Channel 4, 0.002 filter. Channel 4 had the least number of slow frequency cycles. As this paper presents observations from only one patient, statistical data are not presented at this time.

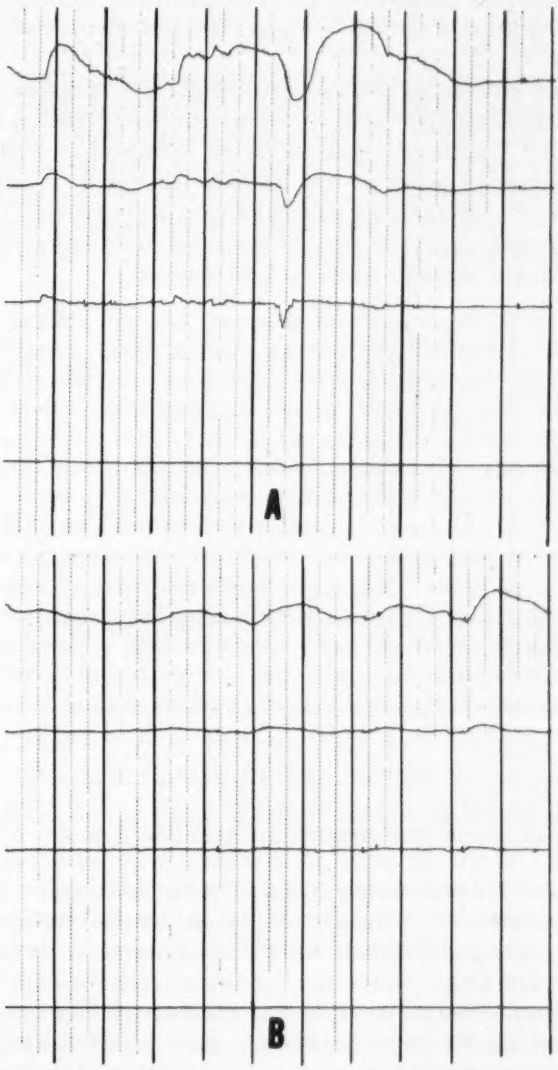

Fig. 5.—*A*, congenital megaloureter. Typical unobserved peristalsis identified by normal peristaltic action potentials. *B*, the same ureter in "resting" period. Curves in Channel 1 represent artifact potentials.

RESULTS

Right Ureter.—Grossly, the markedly dilated right ureter demonstrated normal peristaltic waves, but of decreased rate. The average rate was one peristalsis per minute. The action potentials of these waves were recorded and a representative

tracing is presented in Figure 2. Peristalsis was qualitatively characterized by a triphasic complex; the first and third curves were electropositive and the second curve was electronegative. Quantitatively, these individual curves were of normal duration but of slightly decreased voltage as compared with statistics obtained from other ureters dilated to a comparable degree. Minimal after-potential waves were present.

Interspersed with the above electroureterograms were occasional forceful prolonged peristaltic waves typical of those observed in ureters dilated on an obstructive basis (Fig. 3*A*). Systole was sustained for an average of five seconds prior to diastole. This type of peristalsis was routinely followed by at least one after-potential curve. For comparison, a electroureterogram is presented (Fig. 3*B*) from another patient with marked ureteral dilatation due to ureterovesical obstruction. The same prolonged period of systole is again observed.

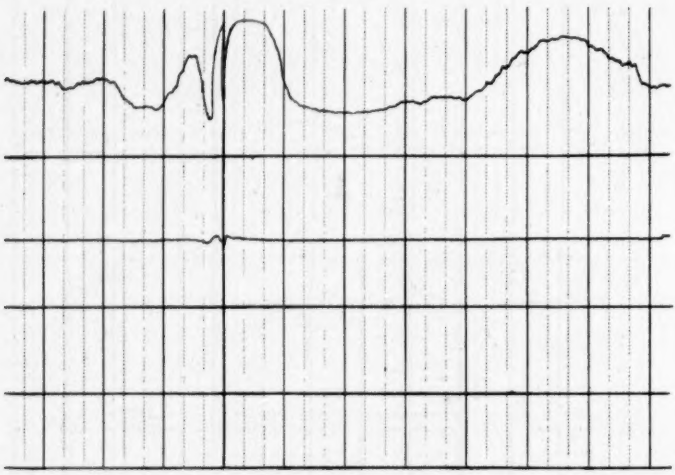

Fig. 6.—Dog. Unobserved peristalsis identified by normal peristaltic action potentials. Cut end of ureter below electrode demonstrated the associated spurt of urine.

Left Ureter.—The tremendously dilated left ureter demonstrated identical qualitative and quantitative characteristics (Fig. 4) as described for the right ureter. Peristalsis appeared grossly normal, but was of approximately the same decreased rate as for the right ureter. Qualitatively, peristaltic action potentials were triphasic and quantitatively, of normal duration. The amplitude (voltage) would have been within normal range for a nondilated ureter, but appeared decreased considering the extreme degree of dilatation present. Only an infrequent after-potential was recorded.

It was observed that typical peristaltic action potentials were occasionally recorded on the graph paper, but no visible peristaltic wave was identified in the ureter (Fig. 5). Associated with this phenomenon, a spurt of urine could be demonstrated coming from the ureter into the bladder. This same observation has been reported previously in dogs (Fig. 6).

COMMENT

The normal ureter at rest is electrically silent. Peristalsis produces depolarization and electrical disturbances termed action potentials. These voltages are subject to measurement. In a previous paper [2] from this laboratory it was reported that the normal electroureterogram is usually characterized by a triphasic action potential. Curves 1 and 3 are electropositive and are separated by Curve 2, which is electronegative. Although the total duration of peristaltic action potentials is remarkably constant, the voltage may vary considerably within a wide range. It has also been demonstrated that the influence of systematic hydration, preoperative medication, anesthetic agent, plane of anesthesia, and respiration do not significantly affect the normal electroureterogram. Electroureterograms obtained in this patient with congenital megaloureter reveal little departure from normal ureteral electrical activity.

Both right and left ureters of the patient were enormously dilated, yet grossly normal peristalsis, but of decreased rate, was observed. Peristaltic action potentials of these dilated ureters were apparently normal. Interspersed with these curves were large moderately forceful peristaltic waves. These curves are typical in both form and duration of the electrical activity obtained in ureters dilated due to obstruction, yet clinically no obstruction was demonstrable. The single distinguishing feature in this patient with megaloureter was a voltage discharge less than might be expected in the normal ureter dilated to the same degree as a result of obstruction. This decreased voltage may be a significant observation in view of the report that these patients have decreased parasympathetic ganglia. It is highly probable, however, that this decreased voltage is attributable to fatigued or failing ureteral musculature produced by marked dilatation and not abnormal parasympathetic innervation. This has been shown to be true in dogs, but no evidence is available at this time to confirm or disprove this impression with regard to humans.

The smooth muscles of the ureter have a double nerve supply; the sympathetic innervation decreases, while the parasympathetic increases, ureteral motility. There are ultimate ramifications of these fibers in the muscle to form a plexus of nonmedullated nerve endings. Not infrequently the ganglion cells from which they arise are also contained in the nerve net near to or among the ureteral smooth muscle cells which they supply. This probably obtains particularly in the lower ureter. To understand the origin and propagation of excitation in the normal ureter, it is necessary to appreciate this intimate structural relationship of the nerve-muscle fiber unit.

The ureteral smooth muscles are composed of fusiform cells connected together by fine intercellular bridges to form a syncytium, by which in all probability the excitatory process can be transmitted from cell to cell. As a consequence, a stimulus applied to part of a sheet of smooth muscle fibers may travel all over the sheet as if it were a single fiber. This phenomenon has been demonstrated in the ureter by Bozler.* He observed that a stimulus applied to any level of the ureter caused a wave of excitation to proceed in both directions away from the stimulus. As a result of this structural pattern, it is difficult to study nerve-muscle relationships in

* References 3, 4, and 5.

normal and abnormal ureters; that is, to distinguish between the effects due to muscle fibers themselves or the nerve and nerve cells which are intimately connected with them. Caution must be exercised, therefore, in interpreting the cause or effects of decreased size and number of parasympathetic ganglia in the bladder or ureter of patients with congenital megaloureter.

It has been established that the ureteral nerve supply is not indispensable for contraction, though capable of causing and modifying it. Engelmann [6] has observed that the ureter requires no extrinsic nerves for contraction. There is evidence that this capacity for automatic contraction originates in the smooth muscle cells themselves, i. e., it is myogenic. The cells in the chick amnion, which are devoid of nerves, have been shown by Ferguson [7] to exhibit these rhythmic contractions. Strips of inner muscular coat of the small intestine, also free from nerve structures, were found to show rhythmic contractions, according to Gunn and Underhill.[8] It is recognized that the smooth muscles of the ureter are very sensitive to mechanical stimuli; distention of the ureter with urine will produce peristalsis. Dye injected into the renal pelvis will pass down the ureter by peristaltic action. Hryntschak † has demonstrated this phenomenon in the absence of parasympathetic innervation by use of the excised kidney-ureter preparation.

The conclusion of Swenson and co-workers that there is a decrease in size and number of parasympathetic ganglia in the bladder, and probably ureters, of patients with congenital megaloureter is not inconsistent, therefore, with the observations now presented of nearly normal propagation of excitation in these ureters. In fact, any alteration in ureteral action potentials is identical to those seen in fatigued or failing ureteral musculature as a result of obstructive lesions.

Perhaps patients with congenital megaloureter have obstructive uropathy that is not clearly demonstrable anatomically, but produces greatly altered physiology as manifest by ureteral dilatation and hydronephrosis. It may be that these children have an extremely small degree of narrowing at the ureterovesical or urethrovesical junctures that is difficult to detect clinically or has become obscure at the time these patients are first examined.

CONCLUSIONS

As a result of the electroureterograms obtained in this patient with congenital megaloureter, it would appear that regardless of the reported anatomical decrease in size and number of parasympathetic ganglia of the bladder, and probably ureter, that electrical silence is not observed. To the contrary, peristaltic activity of these ureters is characterized by a normal triphasic curve of normal duration but probably of a slightly decreased voltage. From the standpoint of electrical activity, these observations are consistent with ureteral changes due to obstruction and not necessarily impaired parasympathetic innervation.

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PNEUMOENCEPHALOGRAPHY IN CHILDREN WITH MENTAL DEFECT AND/OR CEREBRAL PALSY

A Clinicopathologic Study

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AND
BILL GAROUTTE, M.D.
SAN FRANCISCO

IN THE pneumoencephalographic literature, the terms "cortical atrophy" and "cerebral atrophy" are widely used but apparently without regard to the actual pathology. To the radiologist, according to Davidoff and Epstein,[1] cortical atrophy means merely enlarged sulcal markings and cerebral atrophy, enlarged ventricles. These authors admit the inaccuracy of such terminology when applied to congenital malformations of the brain, as a result of which they are confused with the true atrophies. Anderson [2] calls attention to the fact that it is virtually impossible to distinguish between these two types of disorder by means of the pneumoencephalogram.

Our investigation was based on an attempt to find possible x-ray criteria which might be used to differentiate between primary malformations and secondary brain pathology. It seemed to us that the most definitive method of doing this was by correlating pneumoencephalograms obtained during life with postmortem material from the same patients. Very few studies of this sort have been published. Levinson [3] and Brandt, Christensen, and Vesterdal [4] each described one case, while Brody and McAlenney [5] reported 18 cases of cortical atrophy controlled by examination at surgery.

MATERIAL

Thirty brains from patients who died in institutions for the feebleminded were examined.* On all these specimens detailed gross and microscopic pathoanatomic studies were performed. The patients at the time of death ranged in age between 9 months and 20 years. All had had pneumoencephalograms during infancy or childhood. In 24 of the 30 cases, the x-ray films were available for review; in the remaining 6, only the radiologist's interpretation of the films could be obtained.

FINDINGS

The cases were divided into two general groups: Group I consisted of 20 cases of cerebral malformation and Group II of 10 cases showing destructive cerebral lesions as a result of birth trauma, postnatal infection, etc.

From the Laboratory of Neuropathology of the Langley Porter Clinic, and the Departments of Neurology and Anatomy of the University of California School of Medicine.

* Acknowledgment is made to the staffs of Sonoma State Home, and Pacific Colony for cooperating in furnishing the material used in this study.

GROUP I: *Malformations.*—The group of "primary" amentias consisted of (*a*) 17 cases showing various nonspecific cerebral anomalies and (*b*) 3 cases of agyria. Every one of the 20 patients demonstrated mental defect, varying in degree from upper imbecile to lower idiot range. Twelve patients had convulsive seizures, while 11 demonstrated spastic extremities. At least three cases showed definite micro-

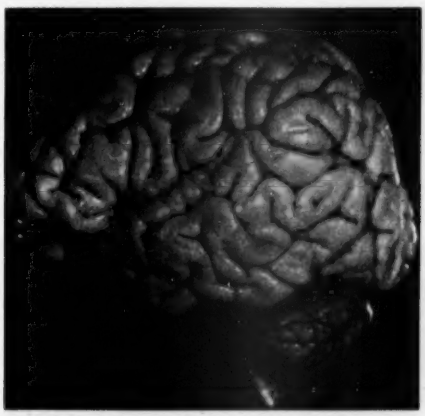

Fig. 1.—Case of nonspecific malformation, with anomalies of convolutional pattern, small occipital and frontal lobes, and enlarged temporal lobes.

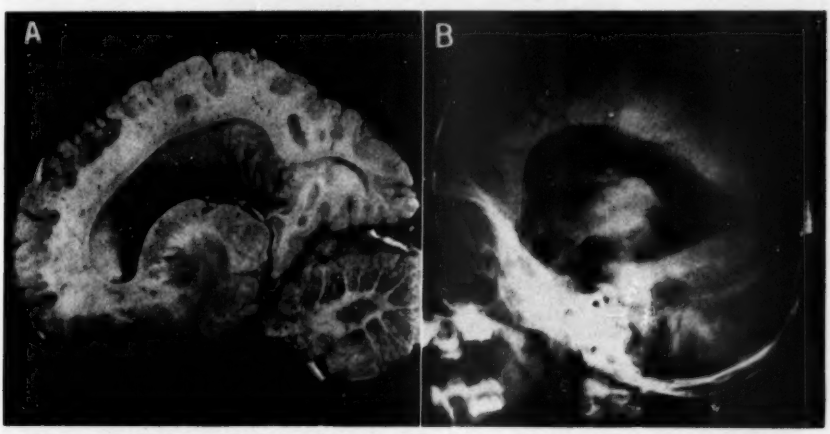

Fig. 2.—Cases of nonspecific malformation. *A*, increased thickness of cortex, reduction of white matter, and enlargement of lateral ventricle. *B*, x-ray changes of flattened occipital region, frontal laking, and dilatation of ventricles.

cephaly. A large percentage of the others were generally underdeveloped so that their heads were smaller than normal, though in proportion with the rest of the body.

(*a*) Nonspecific Malformations: In all cases belonging to this category the essential brain pathology was that of a nonspecific disturbance in the normal pattern of the convolutions and sulci. The sulci were generally shallow and either too many or too few, resulting in ill-defined microgyria, pachygyria, or polygyria. In the

majority, small occipital and frontal lobes contrasted with bulky temporal and parietal lobes (Fig. 1). There was a characteristic increase in relative thickness of the cortex, while the central core of white matter was reduced, resulting in a corresponding dilatation of the ventricles (Fig. 2 *A*). Ventricular enlargement was noted in the pneumograms of the 11 cases where the films were available for review (Fig. 2 *B*). Such ventricular changes in the pneumoencephalogram have been reported by Winkler [6] in 44 of his 78 cases of mental defect.

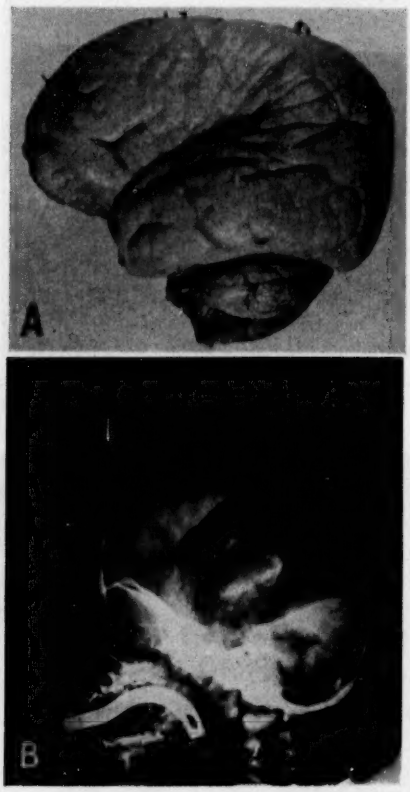

Fig. 3.—Case of agyria. *A*, lack of development of gyri and sulci. *B*, pneumoencephalographic findings of "gas bubbles" in subarachnoid spaces and enlargement of ventricles.

In none of our cases did the radiologist comment on the condition of the subarachnoid spaces, nor were we able to find any pneumographic changes which would correspond to the convolutional anomalies. Nevertheless, there were several features in the x-rays which, in our opinion, appeared to correlate with the pathoanatomic observations. In 7 of 11 cases, there was excessive gas over the frontal poles (Fig. 2 *B*), as illustrated in papers by Crothers, Vogt, and Eley [7] and by Anderson.[2] In six of these seven brains the frontal lobes were distinctly diminished in size. Also, the lateral skull films in most of the cases demonstrated considerable flattening of the occipital region of the skull (Fig. 2 *B*). To study this latter change of shape quantitatively, the "height index" as described by Berry [8] was determined

for all the cases. When compared to Haas's [9] normal data, 5 out of 10 cases of malformation fell outside the normal range, contrasting with the group of cases of acquired pathology, where only 2 out of 10 cases were abnormal. Such skull deformity has been attributed by Schüller [10] and Orley [11] to mechanical factors resulting from the patient's lying constantly on the back of his head. Since in our experience, however, one of the most common features of the malformed brain is the small size of the occipital lobe, we were led to suspect that this phenomenon was the direct cause of the flattening of the occipital region of the skull. Such a possibility has already been implied by Epstein and Davidoff [12] and by Ritvo.[13] Thus, both the laking of gas in the frontal area and the flattening of the occipital bone appeared to us to be of diagnostic significance in cases of cerebral malformation.

(*b*) Agyria: These 3 cases represented extremely severe degrees of malformation. The brains lacked all but the most primitive cerebral fissures and were thus agyric; in every case, there was hydrocephalus ex vacuo (Fig. 3 *A*). The radiolo-

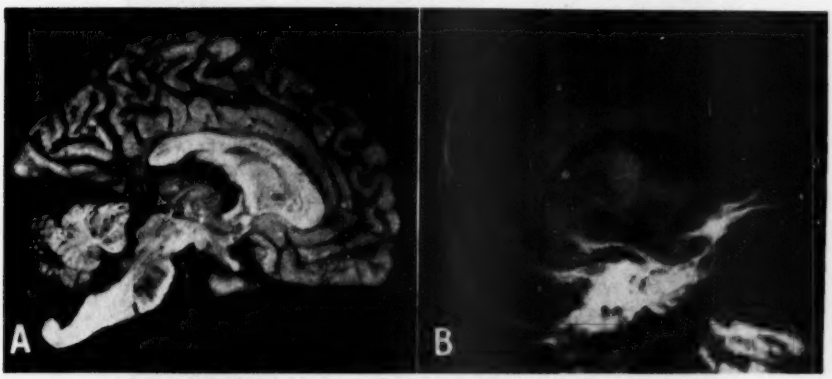

Fig. 4.—Case of cerebral atrophy presumably following birth trauma. *A*, generalized atrophy of convolutions and enlargement of lateral ventricle. *B*, moderate dilatation of ventricle in the pneumoencephalogram.

gist in each instance noted the hydrocephalus and made the diagnoses of "microcephaly vera," "agenesis," and "cerebral atrophy," respectively. The appearance of the subarachnoid spaces was, however, of particular interest in the two cases whose films were available. Very small amounts of gas were noted over the cerebral convexities in both instances, and in one case this gas appeared in the form of bubbles (Fig. 3 *B*). This was not mentioned by the radiologist, possibly because the filling was thought to be subdural rather than subarachnoid. It might be suggested that subarachnoid gas over the smooth convexities of an agyric brain looks a good deal like subdural gas, there being no sulci to cause the normal linear collections.

GROUP II: *Destructive Cerebral Lesions.*—This group of "secondary" amentias included 10 cases in which the pathology was characterized by various types of destructive and obviously acquired lesions. In most instances there was a history of either severe birth trauma or postnatal infection of the central nervous system. The patients in 9 of the 10 cases were mentally defective, the exception being a patient with post-traumatic encephalopathy with convulsions. Eight of the 10 were epileptic; the majority had spastic paralysis of one or more extremities, and 2 were microcephalic.

Fig. 5.—Case of cystic degeneration predominantly of the white matter with history of birth trauma, showing bilateral enlargement of ventricles. *A*, brain. *B*, pneumoencephalogram.

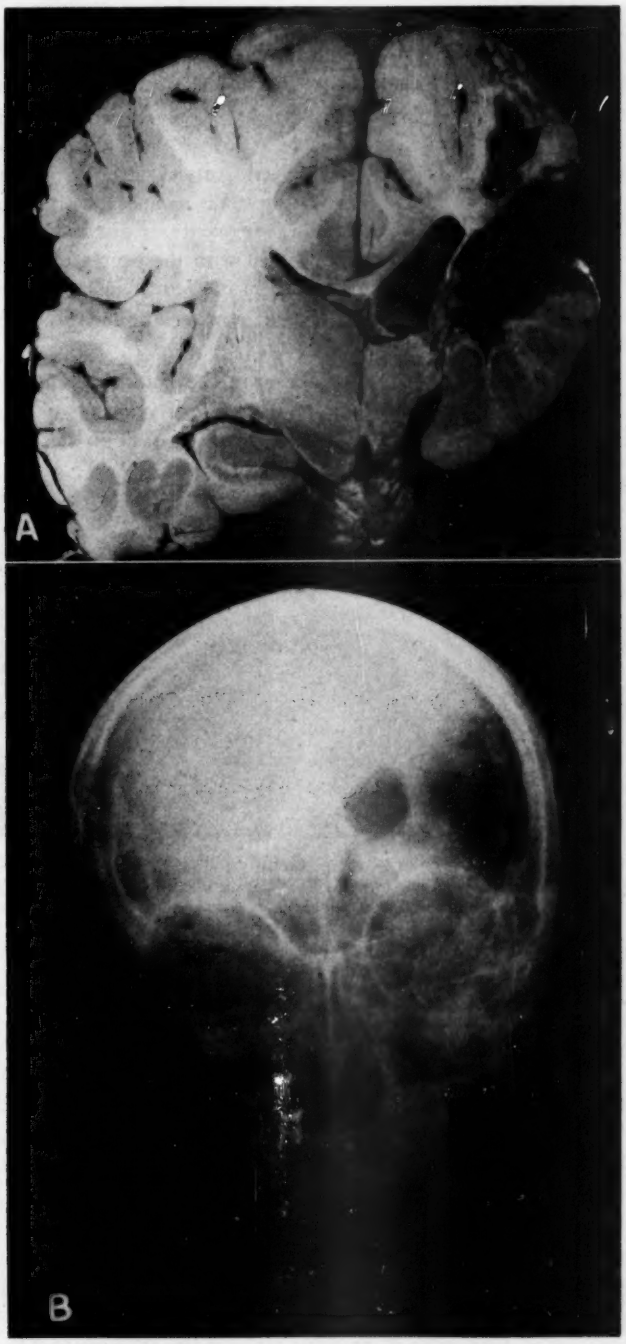

Fig. 6.—Case of cerebral hemiatrophy following scarlet fever (?), showing cystic lesions in left hemisphere with homolateral enlargement of ventricle. *A*, brain. *B*, pneumogram.

From the pathoanatomic standpoint these cases could be classified into four subgroups: (*a*) generalized cerebral and cerebellar atrophy; (*b*) cystic degeneration of white matter; (*c*) cerebral hemiatrophy, and (*d*) primary cerebellar atrophy.

(*a*) General Cerebral and Cerebellar Atrophy: This subgroup was made up of five cases, and the x-ray films were available in four of them. The principal pathologic findings were more or less generalized cortical atrophy associated with secondary degeneration of white matter and, in some instances, with leptomeningeal thickening (Fig. 4 *A*). Examination of the pneumograms disclosed in three cases moderate ventricular dilatation, correlating well with the degree of atrophy of the white matter (Fig. 4 *B*) and normal findings in the fourth case. In two cases, no gas appeared over the convexities, which could very well be the direct result of the leptomeningeal thickening.

(*b*) Cystic Degeneration of White Matter: The marked enlargement of the ventricles (Fig. 5 *A*) in this single case of severe destruction of the white matter contrasted with the more moderate ventricular enlargement in the previous sub-

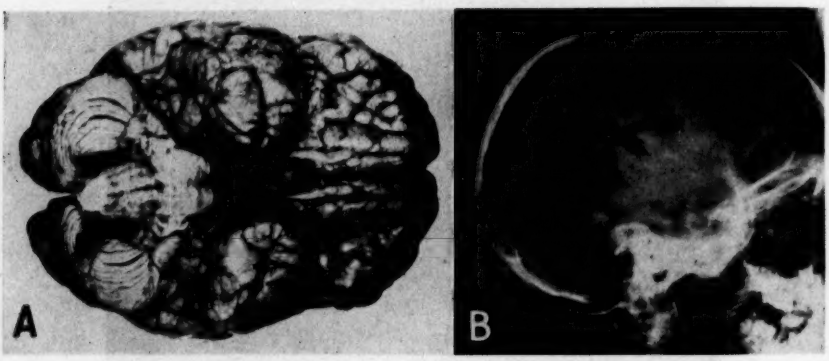

Fig. 7.—Case of primary cerebellar atrophy. *A*, diffuse atrophy of cerebellum with enlargement of cisterna magna. *B*, increased air in region of cerebellum, having the appearance of "lobulation," as seen in pneumogram.

group. Here too, there was a close correlation between the pneumographic and the pathoanatomic findings (Fig. 5 *B*). Absence of gas over the convexities was probably due to thickened meninges, as in the previous subgroup.

(*c*) Cerebral Hemiatrophy: No diagnostic difficulty was experienced with this subgroup, where both the unilateral cystic lesion and the homolateral ventricular enlargement in the two cases showed up clearly in the pneumoencephalogram (Fig. 6 *A* and *B*). The asymmetries of the skull mentioned by Earle, Baldwin, and Penfield [14] and by Dyke, Davidoff, and Masson [15] in cases of hemiatrophy were not observed in the films from these two cases.

(*d*) Cerebellar Atrophy: There were 4 cases of primary cerebellar atrophy, 9 of secondary atrophy, and 1 of congenital hypoplasia of the cerebellum. Satisfactory pneumoencephalographic studies of the region of the posterior fossa were available in three of the primary cases, four of the secondary cases, and in the case of hypoplasia. In all four cases of primary atrophy the cerebellum was uniformly small (Fig. 7 *A*). In one of these cases, lobulation of the cerebellum could be seen

in the lateral x-ray view (Fig. 7 *B*). Murphy and Arana [16] and Orley [11] believe lobulation to be a pathognomonic sign of atrophy, as opposed to hypoplasia of the cerebellum. The pneumogram showed enlargement of the fourth ventricle in three of the primary atrophies and of the cisterna magna in one of these cases. In the fourth case there was an essentially normal x-ray. The one example of hypoplasia of the cerebellum, associated with malformation of the cerebrum, showed a greatly enlarged cisterna magna (Fig. 8 *A* and *B*). It resembled the case reported by Yaskin,[17] who, however, did not have a pathologic study to verify his diagnosis. An enlarged cisterna magna, according to Pancoast, Pendergrass, and Schaeffer,[18] determines the diagnosis of cerebellar atrophy. Our case of hypoplasia clearly controverts this dictum. Considering the findings in our entire series, enlargement of the fourth ventricle and/or cisterna magna was frequently present, irrespective of whether there was primary atrophy, secondary atrophy, or hypoplasia of the cerebellum.

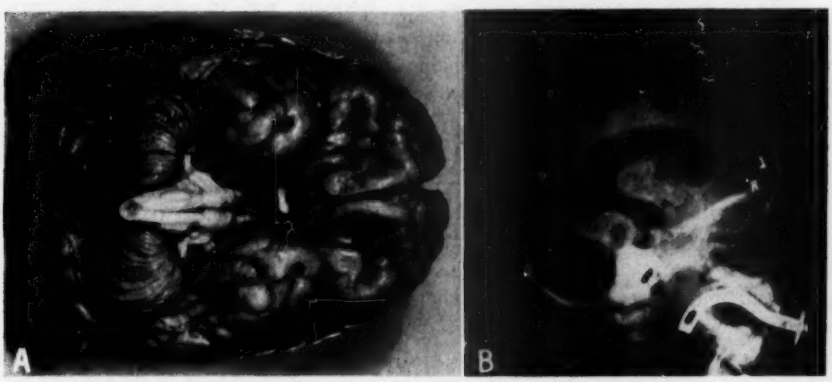

Fig. 8.—Case of cerebellar hypoplasia. *A*, underdevelopment of cerebellum and brain stem and nonspecific malformation of cerebrum. *B*, x-ray changes of marked enlargement of cisterna magna, generalized dilatation of ventricles, and flattening of occipital region of skull.

COMMENT

Casamajor, Laidlaw, and Kozinn [19] feel that there is no adequate reason for doing pneumoencephalographic studies in cases of mental defect, since it usually makes little difference as regards prognosis or therapy. Such a pessimistic point of view does not seem to be justified when applied to a group of neuropsychiatric disorders in which diagnosis, cause, and, consequently, prognosis, are still so poorly understood. In the present state of our knowledge all aids in diagnosis, including the pneumoencephalogram, should be utilized.

From consideration of the present series of cases, a few points emerge, both as regards the group of mental defect and/or cerebral palsy and as regards pneumoencephalography in general.

The flattening of the occipital region of the skull correlates closely with the presence of small malformed occipital lobes of the brain. To our knowledge, this correlation has never before been suggested. It appears to be diagnostically significant for the group of malformations in general.

The presence of frontal laking of gas is possibly of less moment, since, according to Zellweger,[20] the softness and pliancy of the infantile brain makes gaseous laking particularly likely. Nonetheless, its common occurrence in the group of malformations seems related to the underdevelopment of the frontal lobes. In line with our present findings, Orley [11] mentions the occurrence of frontal laking in cases of Mongolian idiocy.

The use of the pneumoencephalogram as an aid in the diagnosis of convolutional anomalies has been disappointing because of the difficulty of obtaining completely adequate visualization of the subarachnoid spaces. Nevertheless, the tendency toward smoothing of the cortex in the malformations is likely to be reflected in the pneumoencephalogram. This was particularly evident in our cases of agyria where the smoothness of the agyric cortex probably accounted for the poor filling of the subarachnoid spaces and for the one case where the gas assumed the form of bubbles. It must be conceded, however, that the chronic arachnoiditis seen in some cases of cortical atrophy may also result in poor filling of the subarachnoid spaces.

The presence of enlarged ventricles is so frequent, both in cases with primary and with secondary pathology, as to take away much of its differential diagnostic value. Severer grades of hydrocephalus would be expected with the severer anomalies as well as with acquired diseases where the white matter is chiefly involved, while with cortical atrophies or minimal malformations, hydrocephalus would be less extreme. The localizing value of focally enlarged distorted ventricles, as in cases of cerebral hemiatrophy, is undisputed.

The diagnosis of cerebellar atrophy as opposed to hypoplasia is strongly suggested by cerebellar "lobulation" in the pneumogram. The presence of enlarged cisterna magna and/or enlarged fourth ventricle does not permit us to draw definite pathoanatomic inferences, since these changes are equally seen in cases of cerebellar atrophy and of hypoplasia.

From a general standpoint, the use of pathologic terms in pneumoencephalographic diagnosis in our opinion should be discouraged. "Cortical atrophy" as used by pneumoencephalographers does not mean the same thing as when used by the pathologist. In the interest of more accurate and understandable terminology, it would appear better to give up such terms as "cortical," "cerebral," or "cerebellar atrophy" and use instead anatomic terms in encephalographic diagnosis.

Pneumoencephalography amounts to in vivo anatomy, giving at little risk to the patient a picture of the brain structure which could otherwise only be obtained on the operating or postmortem tables. By the use of purely anatomic diagnosis, one avoids the pitfall of assuming that ventricular enlargement necessarily means "atrophy of the brain," that subarachnoid widening denotes "cortical atrophy," or that an enlarged cisterna magna or fourth ventricle is diagnostic of "cerebellar atrophy." We are in agreement with Pendergrass,[21] who recommended that the radiologist indicate the various clinicopathologic conditions with which the x-ray findings could be compatible. Used in this way, pneumoencephalography will not become the "treacherous short-cut to diagnosis" that Critchley [22] warned against.

SUMMARY

The pneumoencephalographic and pathoanatomic findings of the central nervous system were compared in 30 cases of mental defect and/or cerebral palsy.

The inadequacy of the terms "cortical," "cerebral," or "cerebellar atrophy" as used by the radiologist is emphasized.

Various diagnostic correlations are drawn between the pneumographic and pathoanatomic findings in the malformations and in the true atrophies.

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CONGENITAL PULMONARY STENOSIS WITH INTACT VENTRICULAR SEPTUM

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CONGENITAL pulmonary stenosis occurs most frequently in conjunction with defect of the ventricular septum, overriding of the aorta, and hypertrophy of the right ventricle. In this syndrome, known as the tetralogy of Fallot, the stenosis practically always involves the infundibulum of the right ventricle, and the pulmonary valve may be normal. In certain instances the stenosis is valvular, but here also the infundibulum shows more or less narrowing.

There is a second type of congenital pulmonary stenosis, less frequent, but by no means rare, in which the ventricular septum is intact. Obviously, there can be no overriding of the aorta. There may, however, be a right-to-left shunt through the foramen ovale if it has remained open or has been forced open by increased pressure on the right side of the heart. The pathology in the region of the pulmonary valve offers a sharp contrast to that seen in the tetralogy of Fallot. First of all, the infundibulum of the right ventricle is distended and shares in the high pressure which occurs in the main cavity of the ventricle. In the second place, the stenosis of the valve is characterized by thickening and fusion of the cusps, so that it appears as a dome-like structure protruding into the lumen of the pulmonary artery. In the third place, the pulmonary artery immediately distal to the valve is dilated.

In both of these forms of pulmonary stenosis, surgery has produced dramatic improvement. The value of the shunt operation by the Blalock or the Potts-Smith technique in the tetralogy of Fallot has been amply demonstrated.

In pulmonary stenosis with intact ventricular septum, pulmonary valvulotomy as developed by Sellors [1] and Brock [2] is the operation of choice. Inasmuch as the infundibulum is normal or distended, the valve can be approached with ease. A shunt operation not only fails to give relief, but actually makes matters worse. The overburdened right ventricle is not relieved, and the increased flow of blood to the left auricle places an additional load on the left heart. Moreover, if the foramen ovale is open, the increased blood entering the left auricle decreases the right-to-left shunt, thus adding to the work of the right auricle and ventricle.

The clinical picture of congenital pulmonary stenosis with intact ventricular septum has been established chiefly from observations on older children and adults.

From the Children's Memorial Hospital.

Of 11 patients operated on by Brock and Campbell [3] in England, none was below the age of 9 years, and five were adults. Adams [4] and co-workers performed right-heart catheterization in 200 persons. Of these, 11 were found to have pulmonary stenosis with open foramen ovale. The youngest patient was 5 years of age, and the majority were older children or adults. Larsson [5] and his co-workers reported 30 cases of pulmonary stenosis diagnosed by cardiac catheterization. The youngest patient was 3¼ years of age. Fifteen were under 10 years of age.

In a personal communication from Dr. Helen B. Taussig, she states that operation has been performed on 81 patients at the Johns Hopkins Hospital with congenital pulmonary stenosis and intact ventricular septum. Of these, two were under 1 year of age, and five were between 1 and 2 years.

We believe that the material which we present in this communication is unusual in that it concerns chiefly infants and young children. The clinical picture in this younger age group differs substantially from that seen in the older patient. We have made observations at the Children's Memorial Hospital on 45 patients believed to have pulmonary stenosis with intact ventricular septum. In 10 patients pulmonary valvulotomy was not done, and in all of these the diagnosis was proved at autopsy. Thirty-five patients were subjected to pulmonary valvulotomy, and in one of these the diagnosis was confirmed at autopsy.

We shall consider first the 10 patients in whom pulmonary valvulotomy was not done.

CASE 1.—S. R., a girl, was born Oct. 5, 1950, and died Oct. 13, 1950. She was admitted to the hospital on Oct. 10, 1950. The baby became cyanotic 24 hours after birth. Oxygen gave temporary relief.

Examination showed slight cyanosis with ashen pallor. The cardiac apex was in the fifth left interspace just outside nipple line. There was a Grade-III systolic murmur, maximal in the fourth left interspace near the sternum. There was no thrill. The second pulmonic sound was normal.

X-ray examination showed the heart to be enlarged, with diminished vascular markings.

Fluoroscopic examination was not done.

The electrocardiogram revealed a right axis shift. There were peaked tall (4 mm.) P waves in Lead II.

The red blood cell count was 4,330,000, with a hemoglobin value of 16.5 gm. per 100 cc.

A severe attack of paroxysmal dyspnea occurred on the day following admission, and the patient grew gradually worse, dying two days later.

Autopsy Findings.—The age at death was 8 days.

The heart weighed 38 gm. (normal, 19 gm.) and showed the following: extreme dilatation and moderate hypertrophy of the right atrium; a defect 11 mm. in diameter in the interatrial septum at the foramen ovale and a fenestrated septum primum; marked stenosis of the tricuspid valve, with thickening of the free margins of the valve; small cavity of the right ventricle, with moderate thickening of the wall; a dome-shaped pulmonary valve with severe stenosis, the lumen measuring 1 mm. in diameter, and marked poststenotic dilatation of the pulmonary artery.

CASE 2.—D. M., a girl, was born Nov. 2, 1949, and died Nov. 11, 1949. She was admitted to the hospital on Nov. 10, 1949. There was cyanosis since birth, increased by crying. The patient was in oxygen during first four days of life.

Examination showed moderate cyanosis at rest. The cardiac apex was in the fourth left interspace at the nipple line. There was a Grade-IV systolic murmur, maximal in the fourth left interspace. A thrill was palpable. The second pulmonic sound was normal or diminished.

X-ray examination showed the heart to be grossly enlarged, with normal or diminished vascular markings.

Fluoroscopic examination was not done.

The electrocardiogram revealed definitely abnormal record. P waves were peaked and tall (5 mm.) in Lead II.

The red blood cell count was 5,790,000, with a hemoglobin value of 16 gm. per 100 cc.

The patient died 30 hours after admission.

Autopsy Findings.—The age at death was 9 days.

The heart weighed 48 gm. (normal, 19 gm.) and showed the following: marked dilatation and hypertrophy of the right atrium; interatrial septum, with opening at the foramen ovale 5 mm. in diameter; mild stenosis of the tricuspid valve, with thickening of the free margins of the valve; extreme hypertrophy of the right ventricle; a dome-shaped pulmonary valve, with severe stenosis, the lumen 1 mm. in diameter, and marked poststenotic dilatation of the pulmonary artery.

Case 3.—J. L., a boy, was born March 16, 1951, and died April 4, 1951. He was admitted to the hospital on March 26, 1951. He had been cyanotic since birth and was constantly in oxygen prior to admission.

Examination showed moderate cyanosis in oxygen. The cardiac apex was in the fourth left interspace at the nipple line. A Grade-II systolic murmur was heard in the third left interspace. There was no thrill. The second pulmonic sound was of normal intensity.

X-ray examination revealed the heart to be boot-shaped. The vascular markings were markedly diminished.

Fluoroscopic examination showed that the heart was not enlarged, and the boot was good. There was a clear pulmonary window. Enlargement of the outflow tract of the right ventricle was found.

The electrocardiogram showed a probably abnormal record. The P waves in Lead II were slightly peaked.

The red blood cell count was 6,450,000 with a hemoglobin value of 20.4 gm. per 100 cc.

A diagnosis of tetralogy of Fallot was made, and an exploratory operation was done. Occlusion of the left pulmonary artery caused immediate and marked slowing of the heart. Anastomosis was not done. The baby died six hours after operation.

Autopsy Findings.—The age at death was 19 days.

The heart weighed 22 gm. (normal, 19 gm.) and showed the following: very marked dilatation of the right artium, with moderate hypertrophy; large defect measuring 9 mm. in diameter in the interatrial septum in the region of the foramen ovale; marked stenosis of the tricuspid valve, with slight thickening of the free margins of the valve; small right ventricle, with thick wall; a dome-shaped pulmonary valve with extreme stenosis, the lumen 1 mm. in diameter, and marked poststenotic dilatation of the pulmonary artery.

Case 4.—D. M., a girl, was born Jan. 7, 1953, and died March 28, 1953. She was admitted to the hospital on March 27, 1953. Cyanosis was noted 24 hours after birth. A heart murmur was heard at that time. Cyanosis persisted. Oxygen was given at home when cyanosis was marked. There was an upper respiratory infection 10 days before admission, with increased cyanosis. An x-ray film at that time was reported to show cardiac enlargement with decreased vascularity of the lungs.

During examination at the time of admission, the baby lay motionless and was deeply cyanotic; respiration was shallow. The hands, feet, and shins were edematous. The heart was enlarged to percussion. A Grade-II-to-III systolic murmur was heard, maximal at third left interspace. The second pulmonic sound was normal. The baby died 15 hours after admission. A work-up was not done.

Autopsy Findings.—The age at death was 11 weeks.

The heart weighed 43 gm. (normal, 23 gm.) and showed the following: enlarged right atrium, with but little hypertrophy; interatrial septum, foramen ovale obliquely patent, probably of functional significance; large tricuspid valve with slight thickening of free margins of valve; extremely enlarged right ventricle, making up about two-thirds of the total heart size; a dome-shaped pulmonary valve with extreme stenosis, the lumen pinpoint in size; moderate poststenotic dilatation of the pulmonary artery with small pulmonary artery distal to dilated portion.

Case 5.—J. F., a boy, was born April 13, 1950, and died Sept. 19, 1950. He was admitted to the hospital on Sept. 16, 1950. The birth was one month premature. The birth weight was 4 lb. 14 oz. (2,200 gm.). The baby was in oxygen during first few days of life and remained in the hospital three weeks. Cyanosis was noted at one month of age. A cardiac murmur was

heard at this time. Cyanosis increased when the bottle was taken. During the preceding week the baby tired easily, took food poorly, and was dyspneic on exertion.

Examination showed good nutritional status and slight cyanosis. There was no clubbing of the fingers. The cardiac apex was in the fourth left interspace at the nipple line. A Grade-III blowing systolic murmur, maximal in third left interspace, was found. There was a faint thrill. The second pulmonic sound was quiet.

X-ray examination showed gross cardiac enlargement with markedly diminished vascularity of the lungs.

Fluoroscopic examination revealed tremendous enlargement of the right ventricle. The right auricular appendage was enlarged. The lung fields were clear.

The electrocardiogram showed right heart strain. There were peaked P waves in Lead II.

The red blood cell count was 3,660,000, with a hemoglobin value of 7.3 gm.

Death occurred suddenly in an attack of paroxysmal dyspnea.

Autopsy Findings.—The age at death was 5 months.

The heart weighed 58 gm. (normal, 29 gm.) and showed the following: an extremely large right atrium, with dilatation and hypertrophy; a large defect 10 mm. in diameter in the interatrial septum at the foramen ovale and fenestration of the septum primum; moderate stenosis of the tricuspid valve, with some thickening of the free margins of valve; moderate hypertrophy of the right ventricle; a dome-shaped pulmonary valve with severe stenosis, the lumen measuring 1 mm. in diameter, and marked poststenotic dilatation of the pulmonary artery.

CASE 6.—R. P., a boy, was born Feb. 27, 1950, and died Sept. 21, 1950. He was admitted to the hospital on Sept. 9, 1950. The baby was cyanotic since birth. There were episodes of paroxysmal dyspnea at 2½ months of age. He was hospitalized elsewhere on two occasions because of spells of difficult breathing. Recently there was constant cyanosis about the lips.

Examination showed slight cyanosis when in oxygen. Respirations were rapid. The cardiac apex was in the fourth left interspace at the nipple line. A Grade-III systolic murmur was found over the precordium. A thrill was palpable. The second pulmonic sound was quiet. Liver pulsations were felt.

X-ray examination showed the heart slightly enlarged. The lung fields were unusually clear. There was a prominent pulmonary artery segment.

Fluoroscopic examination revealed enlargement of the right auricular appendage, inflow and outflow tracts of the right ventricle, and a prominent pulmonary artery segment.

The electrocardiogram showed right heart strain. There were peaked tall (3 mm.) P waves in Lead II.

The red blood cell count was 7,310,000, with a hemoglobin value of 14 gm. per 100 cc.

The patient had repeated attacks of paroxysmal dyspnea and died before he was thought to be in sufficiently good condition to undergo operation.

Autopsy Findings.—The age at death was 7 months.

The heart weighed 90 gm. (normal, 34 gm.) and showed the following: marked dilatation and hypertrophy of the right atrium; a large defect 11 mm. in diameter in the interatrial septum at the foramen ovale, with fenestration of the septum primum; moderate stenosis of the tricuspid valve, with thickening of the free margins of the valve; marked hypertrophy of the right ventricle; a dome-shaped pulmonary valve, with severe stenosis, the lumen 1 mm. in diameter, and severe poststenotic dilatation of the pulmonary artery.

CASE 7.—J. C., a girl, was born Jan. 30, 1949, and died Sept. 22, 1949. She was admitted to the hospital on Aug. 19, 1949. The baby had anorexia and dyspnea for three days and fever, rapid breathing, and cyanosis for 24 hours before admission. Cyanosis was never noted previously.

Examination showed a cyanotic tinge about the mouth. The cardiac apex was at the fourth left interspace outside the nipple line. A harsh blowing Grade-IV systolic murmur was best heard in the fourth left interspace. A thrill was found. The second pulmonic sound was normal. Slight edema of the shins was present.

X-ray examination revealed gross cardiac enlargement, with unusual narrowing of the mediastinum.

On fluoroscopic examination the right auricle and the right and left ventricles appeared markedly enlarged. The left auricle was not prominent on barium swallow.

The electrocardiogram showed right heart strain and a first-degree atrioventricular block. The P waves were peaked and tall (8 mm.) in Lead II.

The red blood cell count was 4,230,000, with a hemoglobin value of 11.3 gm. per 100 cc.

The fever subsided, and signs of congestive heart failure disappeared. The patient was discharged Aug. 19, 1949. She was readmitted to the hospital on Sept. 2, 1949. She had had fever the two days preceding admission.

Examination at this time showed signs of respiratory infection. The patient died three hours after admission.

Autopsy Findings.—The age at death was 8 months.

The heart weighed 114 gm. (normal, 37 gm.) and showed the following: severe dilatation and hypertrophy of the right atrium; anatomical defect 1 mm. in diameter in the interatrial septum at the foramen ovale, probably of very little functional significance; moderate stenosis of the tricuspid valve, with thickening of the free margins of the valve; marked hypertrophy of the right ventricle; a dome-shaped pulmonary valve with a severe stenosis, the lumen 2 mm. in diameter, and severe poststenotic dilatation of the pulmonary artery.

CASE 8.—K. H., a girl, was born March 19, 1952, and died May 5, 1953. She was admitted to the hospital on May 2, 1953. The baby had first been seen in the outpatient cardiac clinic on Oct. 28, 1952. At that time there was a history of slight cyanosis when she cried.

Examination showed a well-nourished baby with slight cyanosis. There was no clubbing of the fingers. A Grade-III systolic murmur maximal in the second and third left interspaces was found. There was a thrill present. The second pulmonic sound was quiet.

X-ray examination revealed a borderline enlargement. There were diminished vascular markings.

Fluoroscopic examination showed a good boot, a good window, and a fair shelf.

The electrocardiogram showed right heart strain. There were peaked P waves in Lead II.

The red blood cell count was 6,870,000, with a hemoglobin value of 11.5 gm. per 100 cc.

The patient was again seen in the outpatient clinic on Feb. 24, 1953. She was gaining and doing well. On admission to hospital on May 2, 1953, she was in extremis with marked dyspnea and grunting respirations. She had been irritable for several days. The baby died three days following admission.

Autopsy Findings.—The age at death was 13½ months.

The Heart weighed 80 gm. (normal, 44 gm.) and showed the following: marked dilatation and hypertrophy of the right atrium; interatrial septum, foramen ovale wide open, with fenestration of the septum primum; relative tricuspid stenosis, with moderate thickening of the free margins of the valve; moderate dilatation and marked hypertrophy of the right ventricle; a dome-shaped pulmonary valve with severe stenosis, the lumen 2 mm. in diameter, also a small thrombosis at the edge of the valve cusp, which may have occluded the lumen, and marked poststenotic dilatation of the pulmonary artery.

CASE 9.—G. M., a boy, was born March 10, 1945, and died Jan. 1, 1950. He was admitted to the hospital on Dec. 31, 1949. Cyanosis was present from birth. The baby was in oxygen during the first three weeks of life. In January, 1949, the first attack of unconsciousness occurred, lasting a half hour. The second attack was in April, the third in November, lasting for two and one-half hours. There were three subsequent attacks.

Examination showed marked cyanosis with suffusion of the eyes and moderate clubbing of fingers. The cardiac apex was in the fifth left interspace, ½ in. (1.3 cm.) outside the nipple line. There was Grade-II systolic murmur, maximal in second left interspace. The heart tones were loud, but the second pulmonic sound was relatively quiet. No thrill was felt. The liver was pulstating, with the edge 1½ in. (3.8 cm.) below the costal margin.

The patient died on Jan. 1, 1950, 36 hours after admission. There was no cardiac work-up.

Autopsy Findings.—The age at death was 4 years 10 months.

The heart weighed 230 gm. (normal, 85 gm.) and showed the following: marked dilatation and hypertrophy of the right atrium; a large defect 12 mm. in diameter in the interatrial septum at the foramen ovale; moderate stenosis of the tricuspid valve, with marked thickening of free

margins of valve; marked hypertrophy of the right ventricle; a dome-shaped pulmonary valve with marked stenosis, the lumen 1 mm. in diameter, and marked poststenotic dilatation of the pulmonary artery, which is small distal to the dilated portion.

CASE 10.—J. L., a boy, was born Aug. 24, 1937, and died March 4, 1948. He was admitted to the hospital on March 2, 1948. Cyanosis had been noted at 4 months of age. Since that time there was increasing cyanosis, weakness, and shortness of breath. Exercise tolerance was very limited. He did not squat when tired.

Examination showed the child to be slender and thin. There was deep cyanosis of the face, fingers, and toes and suffusion of the eyes. Marked clubbing of the fingers and toes was found. The cardiac apex was in the fourth left interspace just outside nipple line. There was a loud harsh systolic murmur, maximal in the fourth left interspace.

X-ray examination revealed a heart shape suggestive of the tetralogy of Fallot but not typical. Vascular markings were much diminished.

Fluoroscopic examination showed a clear pulmonary window. The inflow and outflow tracts of the right ventricle were enlarged.

The electrocardiogram showed right heart strain with peaked very tall (7 mm.) P waves in Lead II.

The red blood cell count was 8,110,000, with a hemoglobin value of 24 gm. per 100 cc.

A diagnosis of the tetralogy of Fallot was made, and a pulmonary aortic anastomosis was done. The patient died several hours after operation because of hemorrhage from an intercostal artery.

Autopsy Findings.—The age at death was 10½ years.

The heart weighed 260 gm. (normal, 119 gm.) and showed the following: marked dilatation and hypertrophy of the right atrium; a defect 10 mm. in diameter in the interatrial septum at the foramen ovale; moderate stenosis of the tricuspid valve, with marked thickening of entire valve, and especially of the free margins; marked hypertrophy of the right ventricle; a dome-shaped pulmonary valve with marked stenosis, the lumen 3 mm. in diameter, and moderate poststenotic dilatation of the pulmonary artery.

An analysis of the protocols listed above gives findings as follows: There was equal distribution of the sexes, five boys and five girls. The age at death in 7 of the 10 patients was less than one year, and of these 3 were under one month of age. Cyanosis was observed in every instance. In one it occurred only terminally, when the patient was thought to have a complicating respiratory infection. It is interesting that in this patient (Case 7) the foramen ovale revealed only a very slight opening which was thought to be functionally unimportant. A precordial murmur was heard in every case. The second pulmonic sound was reported as normal in four, diminished in four, slightly accentuated in one, and not recorded in one. Pulsations of the liver occurred in two. Cardiac enlargement in the x-ray film was the rule. In Case 3, the heart was reported normal in size, and at autopsy the weight of the heart was within normal limits. Decreased vascularity of the lungs was noted without exception. The findings on electrocardiogram were abnormal in all instances and revealed right heart strain in all but the three youngest patients. The P waves in Lead II were peaked and tall, varying from 2 to 8 mm. in height. The red blood cell count was elevated in five, the hemoglobin value above 15 gm. per 100 cc. in four of eight patients for whom blood cell counts were done.

The pathologic findings were so strikingly similar that the same description would have been reasonably accurate if applied to any member of the group. The right auricle was invariably dilated and hypertrophied. The tricuspid valve exhibited a variable degree of stenosis, perhaps only relative in a few instances, due to the increase in size of the right auricle and ventricle. Tricuspid valvular thickening was a constant finding and seemed to be more marked in the two older patients than in the infants. The right ventricle was hypertrophied in all cases.

The most amazing feature was the monotonous uniformity of the architecture of the pulmonary valve. In all cases the valve presented a conical fusion of the three valve cusps, projecting into the pulmonary artery with a tiny opening at the summit of the cone. There was likewise in every instance poststenotic dilatation of the pulmonary artery, usually of marked degree.

We shall now consider the 35 patients in whom pulmonary valvulotomy was done. Their ages varied from 23 days to 15 years. Eight were under the age of one year at the time of operation, the exact ages being as follows: 23 days, 40 days, 2 months, 5 months, 7 months, 8 months, 9½ months, and 10 months. Six were between 1 and 2 years of age, 9 were between 2 and 5 years, and the remaining 12 were between 5 and 15 years. There were 13 boys and 22 girls. Cyanosis was present in 28 of the 35 patients. In 11 it was noted during the neonatal period. In the seven instances in which no cyanosis was noted, all except one patient were beyond the age of 3 years. In the infants, there was usually a history of rapid respiration, frequently with more or less dyspnea. Severe paroxysmal dypsnea occurred in three. In the children who had begun to walk, all except three gave a history of diminished exercise tolerance. Only three patients squatted to rest when tired.

On physical examination, the heart was usually found to be more or less enlarged. A systolic murmur over the precordium was heard in every instance. The murmur varied from Grade II to Grade IV in intensity and was maximal in the second or third left interspace. A thrill was palpated in 29 patients. The second pulmonic sound was described as quiet in 25, normal in 9, and slightly accentuated in 1. Pulsations of the liver were present in 14.

The electrocardiogram revealed a right heart strain in all except four cases. High peaked P waves seen particularly in Lead II were an almost constant feature.

The x-ray study was perhaps the most important feature of the cardiac examination. The anteroposterior x-ray film of the chest revealed cardiac enlargement in all except seven patients, and the heart was usually somewhat globular in outline. In seven, the cardiac enlargement was extreme. The vascularity of the lung fields appeared diminished or normal in all instances.

The fluoroscopic examination revealed enlargement of the pulmonary artery segment in the anteroposterior or right oblique view in 30. When the heart size was increased, the enlargement was found to involve the right auricle and the right ventricle. In none was the left ventricle found to be enlarged. The barium swallow did not reveal a single example of left auricular enlargement. In all cases the aortic arch was found to be on the left. The pulmonary window was usually obscured by the poststenotic dilatation of the pulmonary artery.

An angiocardiogram was done in three cases to rule out the possibility of an overriding aorta. Cardiac catheterization was performed in five cases, confirming the presence of hypertension in the right ventricle with low pressure in the pulmonary artery.

DIAGNOSIS

The findings as recorded in the 45 cases which we have studied outline the features upon which a diagnosis of pulmonary stenosis with intact ventricular septum rests.

The symptomatology is variable, depending upon the severity of the pulmonary stenosis and the presence and degree of shunting of blood from right to left through an open foramen ovale. In the severer cases, cyanosis may occur in early infancy together with attacks of paroxysmal dyspnea. In the milder cases, cyanosis may be minimal or absent, and little or no decrease in exercise tolerance may be noted.

The physical examination of the heart reveals some cardiac enlargement in most instances, a systolic murmur of variable intensity best heard at the base of the heart, usually accompanied by a thrill, and a second pulmonic sound which is quiet or of normal intensity. Pulsation of the liver occurs in a fair percentage of cases, especially in older children.

The x-ray film and fluoroscopic examination usually reveal cardiac enlargement involving the right auricle and the right ventricle. The pulmonary artery segment is prominent. The lung fields are abnormally clear. The electrocardiogram shows right heart strain with tall peaked P waves, particularly noticeable in Lead II.

DIFFERENTIAL DIAGNOSIS

Pulmonary stenosis with intact ventricular septum must be differentiated from other types of pulmonary stenosis, namely, the tetralogy of Fallot, congenital tricuspid atresia, transposition of the great vessels with pulmonary stenosis, and Ebstein's anomaly of the tricuspid valve.

Tetralogy of Fallot.—This is the most frequent congenital cardiac malformation in which cyanosis is present. In the tetralogy, the heart is normal in size or is only slightly enlarged. The x-ray film and the fluoroscopic study show a boot-shaped heart, a concavity in the region of the pulmonary artery segment, a clear pulmonary window in the left antero-oblique view, and a shelf in the right antero-oblique position. The angiocardiogram reveals early entrance of dye into the aorta, and cardiac catheterization may permit entrance of the catheter into the overriding aorta.

Tricuspid Atresia.—The shape of the heart differs from that in pulmonary stenosis with closed ventricular septum, inasmuch as the left ventricle rather than the right ventricle is enlarged, and the heart as a whole shows little or no enlargement. Left heart strain in the electrocardiogram usually serves to make the differential diagnosis.

Transposition of the Great Vessels with Pulmonary Stenosis.—The mediastinum is narrow in the anteroposterior x-ray film. The pulmonary artery is not visualized in its usual position. In doubtful cases cardiac catheterization or angiocardiography may be necessary to establish the diagnosis.

Cor pulmonale may occasionally cause confusion. In this condition, the second pulmonic sound is usually accentuated. Cardiac catheterization reveals increased pressure in the pulmonary artery.

Ebstein's Anomaly of the Tricuspid Valve.—The general picture in this condition closely resembles that seen in pulmonary stenosis with intact ventricular septum. The murmur encountered in this condition is apt to be short, scratchy, or otherwise atypical. In the two cases which we have seen and in others reported in the literature the electrocardiogram has shown right bundle-branch block, but not right heart strain. We believe this to be an important differential point.

PROGNOSIS

Congenital pulmonary stenosis may be so mild that there are no symptoms and no signs that indicate its presence. In such cases, the diagnosis can be established only by cardiac catheterization. In other instances, the narrowing of the pulmonary valve is so extreme that life can be maintained for only a few weeks or months unless the condition is surgically relieved.

SURGICAL TREATMENT

In performing a pulmonary valvulotomy, a curved incision is made beneath the left breast, and the chest is opened through the third interspace. The second, third, and fourth ribs are cut at the costosternal junction. The pericardial sac is opened by a long incision anterior to and parallel with the phrenic nerve.

In the typical case of pure pulmonary stenosis, a few characteristic findings at once present themselves. The pulmonary artery is greatly dilated but slightly constricted at the base of the heart. The right ventricle is full and tense. Palpation over the pulmonary artery transmits to the finger a typical thrill. Actually, one can feel the blood spurting through the constricted pulmonary valve as if it were being forced through a syringe. At times one can feel the small dome-like deformity of the pulmonary valve protruding into the lumen of the pulmonary artery. Increased intracardiac pressure is palpable over the entire right ventricle, and markedly decreased intraluminal pressure is felt over the pulmonary artery.

When such typical diagnostic findings present themselves in a patient who is in good or fair condition, the constricted valve is opened transventricularly with an expanding valvulotome and dilator according to a technique previously described.[6]

If the patient, especially an infant, is in precarious condition and in immediate danger of cardiac anoxia, the small valvulotome (essentially a cataract knife) originally described,[7] is quickly thrust through the wall of the right ventricle and through the constricted valve. The increased opening allows more blood to flow to the lungs, and as the heartbeat consequently improves one may proceed more leisurely with the expanding valvulotome and dilator.

Of the 35 patients who have been subjected to pulmonary valvulotomy, there has been one death. This patient was first seen by us at the age of 11 months. We were uncertain as to whether he had a tetralogy or a pulmonary stenosis with intact ventricular septum. He was deeply cyanotic. The red blood cell count was 8,620,000 per cubic millimeter, and the hemoglobin value was 16 gm. per 100 cc. An angiocardiogram revealed a shunt from the right to the left auricle together with delayed emptying of the right ventricle. One week following the angiocardiogram a cerebral accident occurred producing hemiplegia. The consulting neurologist recommended that operation be postponed for six months. However, the patient's condition grew steadily worse, cyanosis deepened, and he came to operation three months after the onset of the hemiplegia. Death occurred before completion of the operation. The postmortem findings confirmed the diagnosis of valvular pulmonary stenosis with intact ventricular septum. The foramen ovale was open.

In the 34 patients who survived the operation, the results have been most gratifying. Perhaps no cardiac operation is more spectacular. The moment the

pulmonary valve is opened the child becomes pink, and the heartbeat, which is often irregular and slow due to hypoxia and manipulation, picks up a normal rhythm. Convalescence is easy and uneventful.

Observation over the three-and-one-half-year period since this work was begun shows no falling away from the remarkable improvement which occurs immediately after operation. These patients have practically a normal exercise tolerance, and the mothers state that they seem well in every way.

Following operation, we have found that the systolic murmur previously heard remains but is usually less loud and harsh than before operation. A short diastolic murmur has been heard postoperatively in three instances.

It is obviously too soon to assess the long-term results of pulmonary valvulotomy. Whether scarring will cause eventual narrowing of the pulmonary valve or whether significant pulmonary insufficiency will ensue we do not yet know. Our experience thus far leads us to believe that these patients have been permanently rehabilitated.

COMMENT

The routine description of the cases of pulmonary stenosis with intact ventricular septum as set forth above fails to throw into relief certain features which we consider worthy of emphasis. First of all is the fulminating nature of the severer cases, death occurring more or less suddenly in the first weeks or months of life. We have found little reference in the literature to this early fatal form, with the exception of a report by Johnson and Johnson [8] of five fatal cases in patients between the ages of 3 and 19 months.

Careful and repeated observation is essential when one suspects the presence of pulmonary stenosis with closed ventricular septum. This is important, because in the earlier stages this syndrome may be confused with the tetralogy of Fallot. Cyanosis may be present from birth. The early findings on the x-ray film may be indistinguishable from those of the tetralogy. A case in point was that of D. J. Figure 1*A* shows a film taken at the age of 7 months, Figure 1*B*, a year later, just before operation, and Figure 1*C*, two months following pulmonary valvulotomy. The first film could easily pass for a tetralogy. Figure 1*B* shows the tremendous enlargement and characteristic contour which is seen in the extreme cases. Figure 2*A* shows the x-ray film of the patient in Case 5 (J. F.) at the age of 1 month, Figure 2*B*, at the age of 5 months, just a few days before his death.

As a matter of fact, the close resemblance to the tetralogy of Fallot in the earlier stages of pulmonary stenosis with closed ventricular septum may lead to a shunt operation with fatal results. A shunt operation was undertaken on the patient in Case 3 (J. L.) at the age of 19 days. He was cyanotic, a precordial systolic murmur was heard, the heart was boot-shaped, there was a clear pulmonary window, and the pulmonary vascular markings were diminished. When the left pulmonary artery was occluded preparatory to an aortic pulmonary anastomosis, there was immediate marked slowing of the heart. The anastomosis was not done. Death occurred six hours after operation. We have every reason to believe that the baby would have survived a pulmonary valvulotomy had we made a correct diagnosis.

Another patient (Case 10, J. L.) was operated upon for tetralogy of Fallot. He succumbed to a postoperative hemorrhage. He had the typical picture of pulmonary stenosis with closed ventricular septum, but we were not familiar with it at the time. This mistake occurred in a third patient, though with more fortunate

results. A year-old girl was thought to have a tetralogy of Fallot. Her findings seemed fairly typical, except that her heart was larger than is usually encountered in this condition. An aortic pulmonary anastomosis was done. She seemed temporarily improved. Her cyanosis diminished, and she became more active. However, after some months she became dyspneic, her exercise tolerance was diminished, and congestive heart failure occurred. In the meantime her heart had become tremendously enlarged, with a prominent pulmonary artery segment, and her liver was pulsating. At a second operation the anastomosis was occluded, and pulmonary valvulotomy was done. She was immediately relieved, and the parents report that she is now able to keep up with other children. It has been three and one-half years since her second operation.

It is thus seen that we have had difficulty in a few cases in distinguishing between the tetralogy of Fallot and pulmonary stenosis with closed ventricular septum. Even when angiocardiography and cardiac catheterization were employed, the answer was not always clear. In three patients thought to have pulmonary

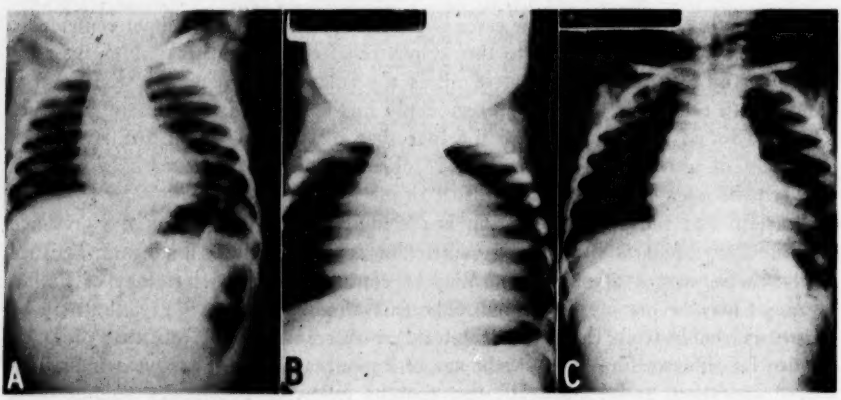

Fig. 1 (D. J.).—*A*, heart at 7 months of age. *B*, heart at 20 months, immediately before operation. *C*, heart two months after operation.

stenosis with intact ventricular septum, it was obvious when the pericardial sac was opened that we were dealing with an infundibular stenosis. The pulmonary artery was large, but the thrill was most intense over the ventricle, and the upper part of the outflow tract was soft in comparison with that of the right ventricle proper. In each instance, the incision in the chest wall was extended laterally, and a shunt operation was done.

Contrariwise, an original posterolateral approach has occasionally been made in anticipation of performing an aortic pulmonary anastomosis for a tetralogy. When there was any doubt about the diagnosis, the pericardial sac was opened. If, then, a pure pulmonary stenosis was found, the valvulotome was introduced through the wall of the pulmonary artery and guided to the stenotic valve.

We believe that a child who has a disabling pulmonary stenosis is entitled to operative relief. Hence, we recommend operation, and, in the doubtful case, undertake to determine the proper procedure after the chest has been opened.

On the other side of the ledger is the patient who has no symptoms and in whom the diagnosis must be made on the basis of physical signs, the electrocardio-

gram, and cardiac catheterization. We did a pulmonary valvulotomy in one child at the age of 4¾ years who was sent to us because of a heart murmur. She had a Grade-III murmur at the base of the heart, a thrill, and a quiet second pulmonic sound. The findings on the x-ray film and the fluoroscopic examination were within normal limits. The electrocardiogram showed right heart strain, though the P waves were not prominent. The mother insisted that the child was perfectly well and able to keep up with other children. No cyanosis had ever been observed, even on hard playing. Yet cardiac catheterization revealed a systolic pressure of 102 mm. of Hg in the right ventricle and 10 mm. of Hg in the pulmonary artery. The decision for operation rested alone on the findings on catheterization.

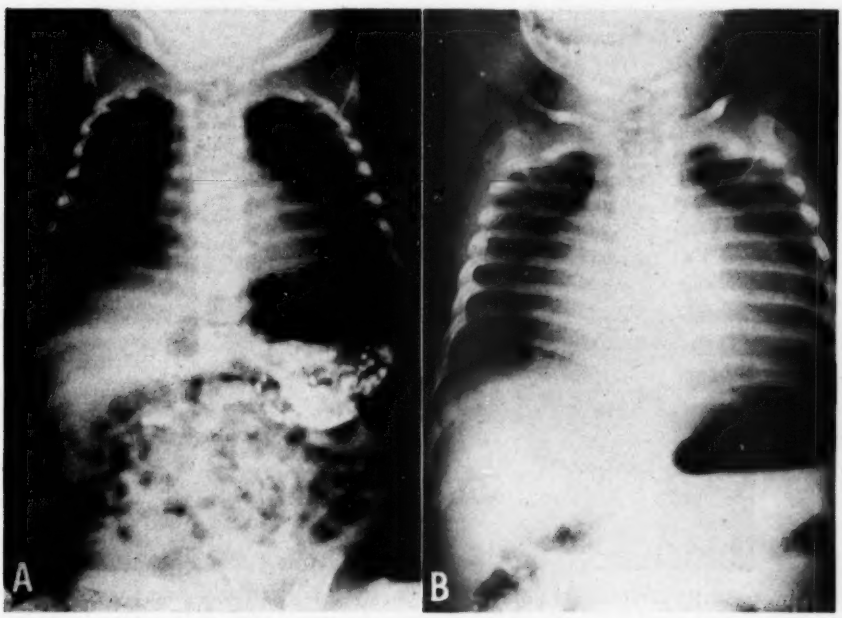

Fig. 2 (J. F.).—*A*, heart at 2 months of age. *B*, heart at 5 months, shortly before death.

One other patient, a sturdy girl of 15 years, also insisted that she could exercise freely without undue fatigue, though she had been restricted somewhat by her physician and by her parents. She had the usual findings of pulmonary stenosis with intact ventricular septum. Cardiac catheterization revealed a systolic pressure of 155 mm. of Hg in the right ventricle and 12 mm. of Hg in the pulmonary artery. Yet in this instance her heart showed no enlargement (Fig. 3).

It is thus seen that some of our patients have been in extremis with marked cyanosis and severe dyspnea, whereas others are practically free of symptoms. This brings us to an explanation of the choice of title for our paper. In severe pulmonary stenosis in which blood is unable to escape through an overriding aorta, the pressure in the right ventricle becomes very high, the tricuspid valve becomes insufficient, and pressure in the right auricle is increased. This may be sufficient to prevent closure of the foramen ovale or to force it open if it has already closed. A right-

to-left shunt is thus established, and if a sufficient amount of blood flows through the foramen ovale, cyanosis results. The presence of cyanosis in the vast majority of our cases leaves no doubt that the foramen ovale was open. For these a proper title would have been "Congenital Pulmonary Stenosis with Open Foramen Ovale." In these few, however, who were free from cyanosis, there was little if any right-to-left shunt. As a matter of fact, the question of whether the foramen ovale is open or closed is a minor consideration. The condition of the ventricular septum is all important. A shunt operation in a patient with a closed ventricular septum leads to his eventual death unless the mistake is discovered and corrective surgery instituted.

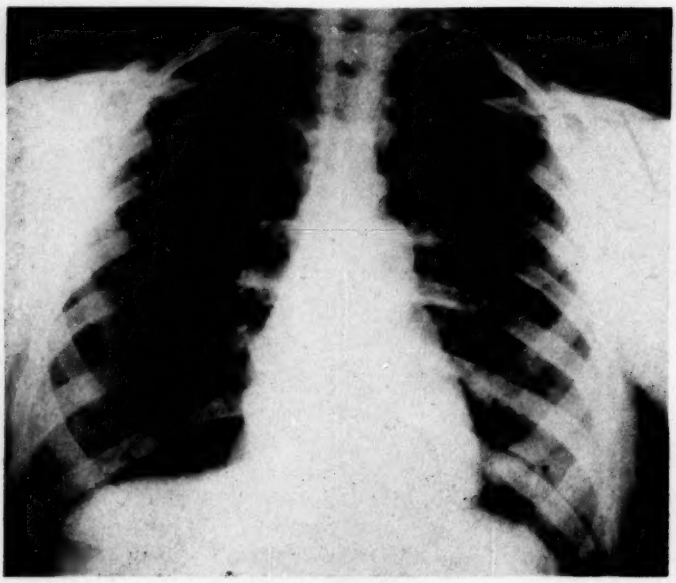

Fig. 3 (R. F.).—15-year-old girl with no cyanosis and good exercise tolerance. Systolic pressure in right ventricle 155 mm. of Hg, in pulmonary artery 12 mm. of Hg. Arterial oxygen saturation 93.5%.

Inasmuch as we have reported the cases of 10 patients coming to autopsy without pulmonary valvulotomy, the casual reader may suspect that we have selected only the more favorable cases for operation. Such is not the case. Three died before we had become acquainted with the clinical picture. Three were in extremis on entering the hospital and died before a cardiac work up was completed. One, 19 days old, was explored in the belief that we were dealing with a tetralogy of Fallot. The remaining three died while we were making the effort to get them in better condition for operation. Our experience has taught us that once these patients begin to go down hill, the end may come suddenly. One died in his first attack of proxysmal dyspnea. Hence, we now employ oxygen, digitalis, and morphine for only a brief period before surgery. In at least three of our infants, congestive heart failure was present at the time of operation. Immediately after the valvulotomy was done, and there was dramatic improvement. Such patients represent surgical emergencies.

SUMMARY

Forty-five cases of pulmonary stenosis with intact ventricular septum have been reported. Ten patients died, in none of whom pulmonary valvulotomy was done. Postmortem studies were made in all. Thirty-five patients were submitted to pulmonary valvulotomy, with one death.

The pathology, symptoms and signs, and laboratory findings are discussed.

The majority of our patients were infants and young children. The fulminating nature of extreme pulmonary stenosis in infancy is emphasized, in some instances constituting a surgical emergency.

The surgical approach is briefly discussed, and the spectacular improvement following surgery is pointed out.

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UVEITIS IN CHILDREN

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UVEITIS in children, though relatively uncommon, may be a very distressing problem. The literature on the subject is scant and for the most part incidental to reports concerned with toxoplasmosis,* sarcoidosis,[6] or juvenile rheumatoid arthritis (Still's disease).† The only series of children with uveitis was reported by Thomson [13] (1928), whose 10 cases had in common a chronic course, uveal tract disturbance without visible fundus change or iritis, and unilateral involvement; apparently the etiology was unknown. Ravin [14] (1944) described a case of iritis following measles in a 3-year-old girl; Hallett [15] reported a case of iridocyclitis coincident with chickenpox in a 7-year-old boy, and Wilder [16] (1950) reported the finding of nematode larvae in the granulomatous lesions of a series of eyes enucleated from children.

The purpose of the present study was to analyze all the cases of childhood uveitis seen in connection with the uveitis survey being conducted by the staff of the Francis I. Proctor Foundation for Research in Ophthalmology and the Department of Ophthalmology, University of California Medical Center. A total of 810 cases of uveitis have been surveyed in a five-and-a-half-year period, and 47 of them, or 5.8%, were in patients under 16 years of age. This figure is unquestionably higher than the general average which is probably closer to 2 or 3%. Blegvad [17] (1941) found in Denmark that 2.2% of 896 cases of uveitis were in children under 15 years of age. The higher percentage in our series was probably due to the special interest of one of us (M. J. H.) in toxoplasmosis; many cases of healed chorioretinitis were referred for study which might otherwise have been ignored.

The records of all cases were drawn from the files of the uveitis clinic, to which both clinic and private patients are referred. They were studied as outpatients or hospital patients, and each was examined by at least one of us. Since many cases were referred because of suspected toxoplasmosis, and most other cases for diagnostic opinion because of recurrence, unusually severe, and/or special therapeutic problems, the series must be regarded as highly selected and unsuited to statistical

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† References 7, 8, 9, 10, 11, and 12.

analysis. Our evaluation of certain survey procedures changed during the five and a half years, moreover, so that some of the tests applied early in the study were discarded and others were initiated.

All the records contained the following data: (1) brief medical history and detailed ophthalmic history; (2) complete physical examination; (3) detailed examination of the eyes, including slit-lamp biomicroscopy and funduscopy with pupils dilated; (4) skin tests, including the tuberculin, Frei, toxoplasmin, coccidioidin, histoplasmin, and Brucellergin tests; (5) serologic tests, including the Toxoplasma dye test, the Toxoplasma complement fixation test, Brucella agglutination and complement fixation tests, Q-fever complement fixation test, and the Kline and Kahn test for syphilis; (6) neutralization test for toxoplasmosis; (7) white blood cell count and differential count; (8) sedimentation rate, and (9) roentgen chest examination

TABLE 1.—*Etiologic Diagnosis or Disease Association in Anterior Uveitis (Eighteen Cases)*

Diagnosis or Association	No. of Cases	Age at Onset, Yr.
Heterochromic iridocyclitis	1	13
Boeck's sarcoidosis	1	11
Syphilitic keratouveitis	1	6
Traumatic iritis	1	8
Brucella keratouveitis	1	14
Juvenile rheumatoid iritis	1	1½
Unknown	12	

TABLE 2.—*Types of Anterior Uveitis of Unknown Etiology (Twelve Cases)*

Type	No. of Cases
Iridocyclitis, nongranulomatous	5
Iridocyclitis, granulomatous	3
Cyclitis	4

and special films of joints, skull, and phalanges when indicated by the history or physical examination.

The patient's age at the onset of the uveitis was determined whenever possible. In many of the cases with healed chorioretinitis it was impossible to state when the lesions had first occurred. In cases in which there was a healed macular lesion with a squint it seemed logical to assume that the lesion had been present at birth or soon thereafter.

ANALYSIS OF CASES

There were 47 children under 16 years of age in our total series of 810 cases of uveitis. Their cases could be divided into those of anterior and those of posterior uveitis, depending on which portion of the uvea was involved primarily, and there were more than half again as many cases of posterior uveitis (29) as there were cases of anterior uveitis (18). Curiously enough, there were no instances of generalized uveitis among the children.

Anterior Uveitis (18 cases).—An etiologic diagnosis, definite or presumptive, or a specific disease association was determined in 6 of the 18 cases of anterior uveitis. The 12 cases in which we were unable to determine the etiology or any specific disease association could be typed as shown in Table 2. The findings in

these 12 cases are elaborated in Tables 3, 4, and 5. Table 3 shows that four of the five cases of nongranulomatous iridocyclitis were acute and that the fifth was subacute. Three of the five patients had definite allergic diatheses. In one of these (a 12-year-old girl, M. M.) the eyes cleared simultaneously with the removal of a pet cat. Table 4 shows that the three cases of granulomatous iridocyclitis were chronic. One of the patients (R. S.) had one painful eye enucleated, and the histological studies showed only a nonspecific type of chronic inflammation. Cultural studies were not made. The four cases summarized in Table 5 were listed as instan-

TABLE 3.—*Iridocyclitis, Nongranulomatous Type (Five Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
D. C.	M	6	O. U.	—	Acute iridocyclitis; allergic diathesis
B. K.	F	5	O. U.	—	Acute iridocyclitis; allergic diathesis
J. W.	M	10	O. U.	—	Acute iridocyclitis, recurrent
A. E.	M	10	O. U.	—	Subacute iridocyclitis
M. M.	F	12	O. U.	—	Acute iridocyclitis; allergic diathesis

TABLE 4.—*Iridocyclitis, Granulomatous Type (Three Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
R. S.	F	7	O. U.	—	Chronic iridocyclitis; band keratitis
M. D.	F	14	O. D.	—	Chronic iridocyclitis; allergic diathesis
P. B.	F	8	O. U.	—	Chronic iridocyclitis; eosinophilia, 2-19%

TABLE 5.—*Cyclitis (Four Cases)*

Patient	Sex	Age at Onset, Yr.	Eye(s) Involved	Skin Test and Serology	Positive Findings
L. H.	F	11	O. U.	—	Allergy; crystalline deposits on irises
W. N.	M	12	O. U.	Brucella +	Allergic diathesis
G. C.	M	9	O. U.	—	Eosinophilia, 7-10%
M. D.	M	4½	O. U.	1st PPD +	Chronic cyclitis; band keratitis

ces of cyclitis because there was very little sign of reaction in the anterior chamber and no visible chorioretinitis; since in some cases the extreme periphery of the fundus could not be seen, we cannot say that in these there was no peripheral chorioretinitis. Anterior vitreous opacities constituted the most interesting finding in this group. The irises of one patient (L. H.) with a chronic bilateral cyclitis showed crystalline deposits that were thought to be lipoidal in nature.

Posterior Uveitis (29 cases).—A presumptive diagnosis of toxoplasmosis was made in 10 of the 29 cases of chorioretinitis. The significant findings in these and in the 19 undiagnosed cases are summarized in Tables 6, 7, and 8. The chorioretinitis was active in only 7 of the 19 cases that could not be diagnosed etiologically; of the 12 healed cases, 3 were associated with strabismus.

Differential Diagnosis of Uveitis in Children.—In the course of this survey six children were referred to the uveitis clinic who did not in fact have uveitis. Diag-

nostic study showed four of them to have Coats's disease. The ocular involvement was unilateral in all four, and three of the four patients were boys. The disease was definitely exudative in three and hemorrhagic in the fourth. In one case that was

TABLE 6.—*Chorioretinitis Presumed to be Due to Toxoplasmosis (Ten Cases)*

Patient	Age, Yr.	Sex	Toxo Dye Test	Skin Neut. Test	Skull X-Ray	Squint	Mental Retardation	Fundus
S. A.	3	M	.....	+	+	ST	+	O. S. healed
N. H.	8	M	.....	±	+	ST	+	O. U. healed
S. J.	14	F	.....	+	+	XT	+	O. S. active
S. F.	11	F	.....	+	+	XT	—	O. U. healed
S. F.	11	F	.....	+	+	ST	—	O. U. healed
F. S.	2½	M	1:2,048	..	—	ST	?	O. U. healed
G. S.	2½	M	1:256	..	—	ST	?	O. U. healed
L. B.	16	F	—	—	+	ST	+	O. U. healed
M. F.	12	M	1:128	+	+	—	—	O. S. active
L. S.	7	F	1:64	..	+	ST	—	O. U. healed

TABLE 7.—*Active Chorioretinitis of Unknown Etiology (Seven Cases)*

Patient	Age at Onset, Yr.	Sex	Skin Tests	Toxoplasma Dye Test	Fundus Lesions
R. T.	15?	M	All —	0; neut. +	O. U.; O. D. active
B. W.	14?	F	All —	0; neut. —	O. U.; O. D. active
R. S.	12?	M	All —	—	O. D., subacute, elevated
C. E.	6?	F	All —	—	O. S., proliferated
C. P.	5?	F	All —	1:16	O. U.; O. D. active
S. B.	15?	F	1st PPD ± coccidioidin +	—	O. U., peripheral
I. S.	3?	M	Toxo +	1:64	O. S., acute

TABLE 8.—*Healed Chorioretinitis of Unknown Etiology*

Patient	Age, Yr.	Sex	Skin Tests	Toxo, Dye Test	Comments
Y. P.	7	F	All —	—	.....
M. J.	8	F	All —	1:8	.....
P. B.	9	M	All —	1:8	.....
J. W.	12	F	Toxo +	...	Skin neut. test +; toxo?
W. L.	12	M	Toxo +	...	Skin neut. test —
F. F.	9	M	Brucella +	1:128	Toxo?
C. M.	12	F	Brucella and coccidioidin +	1:128	Toxo?
J. G.	10	M	1st PPD +	—	.....
E. B.	6	M	All —	1:16	.....
P. F.	4	F	All —	1:128	XT, age 1½ yr.; toxo?
J. N.	15	F	All —	—	ST, age 1½ yr.
M. S.	14	F	Toxo and histo +	1:16	ST from birth; toxo?

followed for five years the process resulted in a retinal detachment with loss of vision. Certainly, the exudative type of Coats's disease, especially in its early stages, is a condition that must be differentiated from uveitis.

The other two cases that presented a problem in differential diagnosis were examples of heredomacular degeneration without central nervous system involvement. In both of them all tests were negative, and clinically there were no signs of inflammation. The vitreous was clear. In both the disease had developed insidiously, and the lesions were bilateral and symmetrical.

COMMENT

One-third of the cases of anterior uveitis in this series could be given a reasonably definite etiologic diagnosis or could be explained as a manifestation of a specific disease entity. A case of heterochromic iridocyclitis occurred in a 15-year-old girl who had first noted blurring in one eye when she was 13 years old. The iris of the involved eye was lighter in color; the lens showed an early posterior subcapsular cataract. Heterochromic iridocyclitis of Fuchs usually manifests itself early in adult life and is rare in children.

Boeck's sarcoidosis involving the eyes was seen in an 11-year-old Negro girl. Clinically the uveitis was a granulomatous iridocyclitis. X-ray films of the chest showed hilar shadows, and lymph node biopsy findings were consistent with sarcoidosis. This disease is rare in children. Oblath and Farber [18] (1950) reported a series of 40 cases seen here at the University of California Hospital and at the San Francisco Hospital. Among these only three were in patients 15 years of age or younger. In a series of 29 proved cases of sarcoidosis reported by Woods and Guyton [19] (1944), 15, or 52%, were complicated by uveitis, and 2 of the 15 were in patients 15 years of age or younger. Although a definite diagnosis of systemic sarcoidosis can be made on the basis of lymph node and skin biopsies, tuberculin test, etc., the diagnosis of ocular sarcoidosis can be presumptive only, since all cases of the disease do not involve the eye. According to Thornhill and Thornhill,[6] eye involvement is especially rare in children.

Syphilitic keratouveitis was the definite diagnosis of the eye condition of a six-year-old boy with congenital syphilis. Serum tests for syphilis were positive, and the clinical findings were characteristic.

Brucella keratouveitis was the presumptive diagnosis in the case of a male dairy worker who was born and raised on a dairy farm. Raw milk was the only type of milk he had drunk from infancy, and he gave a history of several bouts of unexplained fever associated with malaise. His uveitis apparently dated from his 14th year. The clinical examination of the eyes showed a chronic iridocyclitis with nummular keratitis and early band keratitis. The Brucella skin test was positive, but the blood agglutinin and complement fixation titers were negative. Woods and Guyton [19] mentioned two cases of chronic brucellosis with nummular keratitis.

A case of uveitis presumed to be one of juvenile rheumatoid iritis was seen in an 8-year-old girl who had had arthritis when she was 1½ years old. Clinically the facies was bird-like, and the eyes showed a very chronic, low-grade iridocyclitis, early band keratitis, and complicated cataract. In spite of the current absence of arthritis, a presumptive diagnosis of juvenile rheumatoid iritis seemed to be justified. Blegvad [17] stated that a patient can recover completely from the arthritic element in this syndrome, and Franceschetti and Bamatter [9] pointed out that the typical eye findings of low-grade iridocyclitis, complicated cataract, and band-shaped keratitis are important early manifestations of juvenile rheumatoid arthritis (Still's disease).

No etiologic diagnosis, presumptive or definite, could be made in 12 cases of anterior uveitis. In 7 of the 12 cases there was an allergic diathesis, and 2 other cases showed a constant eosinophilia. Skin tests and serologic tests were consistently negative except in two cases, one with a positive Brucella skin test but a negative serologic test, the other with a positive tuberculin test.

While it is well known that a number of acute febrile diseases, particularly varicella, influenza, measles, and epidemic parotitis, are occasionally followed by uveitis, especially during their convalescent periods, it is noteworthy that none of the cases in this series followed any of the childhood exanthemata.

The infrequency of iridocyclitis in children is unexplained. The recent report of Reinhardt and Li [20] (1953) on the production of arthritis in experimental animals by the injection of the growth factor of the anterior pituitary hormone suggests that iridocyclitis, too, may eventually be explained on an endocrinologic basis, since arthritis and iridocyclitis are closely related clinically.

Healed chorioretinitis is a not uncommon finding in children's fundi. By virtue of our interest in congenital toxoplasmosis, however, we have undoubtedly seen more of it than one would find in a random sampling of cases. A presumptive diagnosis of toxoplasmosis was made in 10 of our children. A positive methylene blue dye test (Sabin and Feldman) and a positive complement fixation test are reliable and essential serologic criteria for the diagnosis of toxoplasmosis. If the child has clinical manifestations of the disease and the serologic tests are confirmatory, the diagnosis may be considered definite. After the age of 5 years, however, the problem becomes much more difficult because of the marked decrease in the patient's reaction to the methylene blue dye test and the reduced titer of complement-fixing antibodies in his serum. In fact, an unequivocal diagnosis is usually impossible in the older age group. Children with toxoplasmosis are usually mentally retarded in addition to having poor vision, and this adds to the importance of making the diagnosis. It is especially vital to the parents, since the chances are very good that subsequent children would be toxoplasmosis-free.

Sabin and Feldman [21] (1949) showed that toxoplasmosis was not the only cause of chorioretinitis and cerebral calcification. They described a syndrome rarely seen in infancy that is characterized by extensive destruction of brain tissue with hydrocephalus, by diffuse cerebral calcification, and by choroidal lesions associated with vascular changes; it can be differentiated from toxoplasmosis only by serologic tests or histopathological examination. There were no cases of this syndrome in our series.

Of the 19 cases of chorioretinitis in which we were unable to arrive at any sort of etiologic diagnosis, only 7 were active. The tuberculin reaction was negative in all seven, and only two showed Toxoplasma dye test titers (1:16 and 1:64 respectively). The chorioretinal lesions were bilateral in all but one case and recurrent in three. The one unilateral case showed a large proliferative choroidal lesion in a 6-year-old girl with a constant eosinophilia of 10%. It suggested a parasitic involvement, but no parasites were found. Since Wilder's (1950) finding of nematodes in histopathological preparations from endophthalmitis, an effort has been made to find the parasites in suspected clinical cases. So far this effort has been unsuccessful

There were 12 cases of healed chorioretinitis of unknown origin, 3 of them with an associated strabismus. In each of these three cases the squinting eye showed a healed macular lesion. In their analysis of 1,000 cases of strabismus, Granström and Magnusson [22] (1949) found 6 to be due to toxoplasmosis. Congenital toxoplasmic infection was suspected in 5 of our 12 undiagnosed cases, but the evidence was insufficient for definitive diagnosis.

Our series did not contain any case of posterior uveitis associated with acute rheumatic fever, although one of us (M. J. H.) has seen in this disease a number of cases of choroidal inflammations resembling miliary tuberculosis. Rudolph,[23] on the other hand, studied a series of 105 cases of rheumatic fever in an Army hospital and found no ocular involvement.

It will be noted that no case in this series could be ascribed definitely to tuberculosis in spite of the widespread belief, particularly among German ophthalmologists, that this infection is the major etiologic factor in chronic forms of uveitis. Thiel,[24] for example, in a statistical review of all cases of endogenous uveitis treated at the Frankfurt University Eye Clinic and Hospital during the years 1935 to 1952, concluded that approximately 75% were tuberculous in origin. It is true that tuberculosis has declined strikingly in Northern California in recent years, but other extrapulmonary manifestations of the disease, particularly phlyctenular keratoconjunctivitis, are still being seen with considerable frequency. It is possible that tuberculosis tends to spare the uveal tract in childhood. One of us (P. T.), in the course of studies conducted with M. H. Fritz on phlyctenulosis among Alaskan Indian and Eskimo children,‡ was struck by the absence of uveitis except as a complication of the keratitis of phlyctenulosis. This type of secondary uveitis is ordinarily transitory and disappears with subsidence of the corneal attack, although, very exceptionally after repeated flare-ups, it may lead to atrophy of the globe. In a personal communication, Dr. Fritz stated that he had not seen a typical case of endogenous uveitis in the course of his examination of more than 2,000 Alaskan native children, in spite of the fact that, according to the results of tuberculin testing and the high incidence of pulmonary and bone and joint tuberculosis, the children were almost universally infected.

Healed pigmented foci are frequently seen in otherwise normal fundi, and there is usually no history of inflammation. Perhaps many of these will prove to be due to a defect in development rather than to an inflammatory process. At present, however, we cannot state that any of the healed chorioretinal lesions we have seen were developmental defects.

SUMMARY AND CONCLUSIONS

In a series of 810 cases of uveitis surveyed at the Francis I. Proctor Foundation for Research in Ophthalmology and the Department of Ophthalmology, University of California Medical Center, 47, or 5.8%, were in children under 16 years of age. Of the 47 cases, 18 were primarily of anterior uveitis and 29 were primarily of posterior uveitis.

Only one-third of the cases of anterior uveitis could be given an etiologic diagnosis, presumptive or definitive, or the condition identified as a manifestation of

‡ References 25, 26, and 27.

a specific disease. Of the 12 undiagnosed cases, the only frequent findings were an allergic diathesis, in 7 cases, and a constant eosinophilia, in 2 others, suggesting that allergy may be an important factor.

Of the 29 cases of posterior uveitis, a presumptive diagnosis of toxoplasmosis was made in 10, and 19 could not be explained. The practical importance of determining the presence or absence of toxoplasmosis is emphasized.

The chorioretinitis was active in only 7 of the 19 cases of posterior uveitis which could not be diagnosed etiologically.

Four cases of Coats's disease and two cases of heredomacular degeneration were referred as instances of uveitis.

Iridocyclitis is extremely rare in children; chorioretinitis is relatively common.

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Case Reports

ESOPHAGEAL ULCER IN VERY YOUNG INFANTS

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ESOPHAGEAL disease constitutes a particularly serious threat to the life and well-being of newborn and very young infants. Inflammation and ulceration of the distal esophagus is rare in healthy infants, but is found not infrequently in postmortem studies of the newborn. The complications of hemorrhage, perforation, or obstruction are almost always fatal, and usually occur before the underlying disease has been recognized. If especially sought for, inflammatory changes will be found frequently in the distal esophagus at postmortem examination of the newborn. The two following cases are thought to be unusual, the first because it was characterized by fatal hemorrhage into the gastrointestinal tract and the second because of the complete obstruction which abated after gastrectomy.

CASE 1.—This 2,050 gm., 46.97 cm. baby girl was born at 5: 30 a. m. on Oct. 17, 1952, after a normal pregnancy and normal delivery. The infant's condition was critical; marked molding of the head was evident. The baby was very limp and had marked asphyxia and was placed in an air lock immediately. While in the air lock, she received ½ ampule of caffeine and sodium benzoate. After five minutes in the air lock, respirations became full and regular, and the infant appeared to be in fair condition. Upon transfer of the infant to the nursery, the color appeared to be poor, and respirations were shallow and irregular, but no retractions were present. The child was placed in an incubator and given humidified oxygen and the routine premature antibiotic and vitamin therapy. She appeared to be improving until 7 p. m. when, having taken a little water by mouth, she suddenly vomited a large amount of bright red blood. Blood continued to ooze from the child's mouth until death. No bleeding point could be found within the pharynx.

Blood cell counts were performed at four-hour intervals. The hemoglobin level fell only slightly, but at 7 a. m. the following morning the child passed a large stool consisting primarily of blood clots. A 35 ml. blood transfusion was given which seemed to improve her somewhat. She nevertheless continued to bleed from her mouth and was listless, pale, and gasping. She died 33 hours after birth.

At postmortem examination all skin surfaces were very pale. Significant findings were confined to the gastrointestinal tract: the esophagus was lined by smooth gray mucosa throughout the upper two-thirds; on the anterior wall of the distal third was a linear ulceration of the mucosa 1.0 cm. by 0.1 cm. The lower pole of the ulcer was just above the gastric cardia. The serosa covering this area was dark red in color, and gelatinous material was seen in the interstitial tissue. The stomach contained some thick mucoid material which was dark green to black in color. The rugae were prominent. When the mucosa was washed, it appeared to be smooth gray-green. The pylorus was a firm muscular ring and was not unusual. Distal to the pylorus the intestinal tract was filled as far as the rectum with dark clotted blood which ranged in color from black to reddish-black. No pathological change was seen in the gastrointestinal tract except that of the esophageal ulceration. The liver and gall bladder were normal, as was the pancreas. The adrenals were normal in size, shape, and appearance.

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Microscopic study of sections from the esophageal ulcer revealed necrosis of the mucosa, but at most points the basement membrane was intact. The submucosa, muscularis, and serosa were infiltrated moderately by inflammatory cells, both neutrophiles and mononuclear cells. There was associated edema of the submucosa and serosa. The muscularis contained foci of fresh hemorrhage underlying the ulcer, but at no point did these communicate with the surface.

Comment.—This is an example of the acute or "stress ulcer" of the esophagus, with hemorrhage into all coats of the esophagus as well as into the lumen. If one excludes the most common cause, ingestion of a corrosive agent, the cases of esoph-

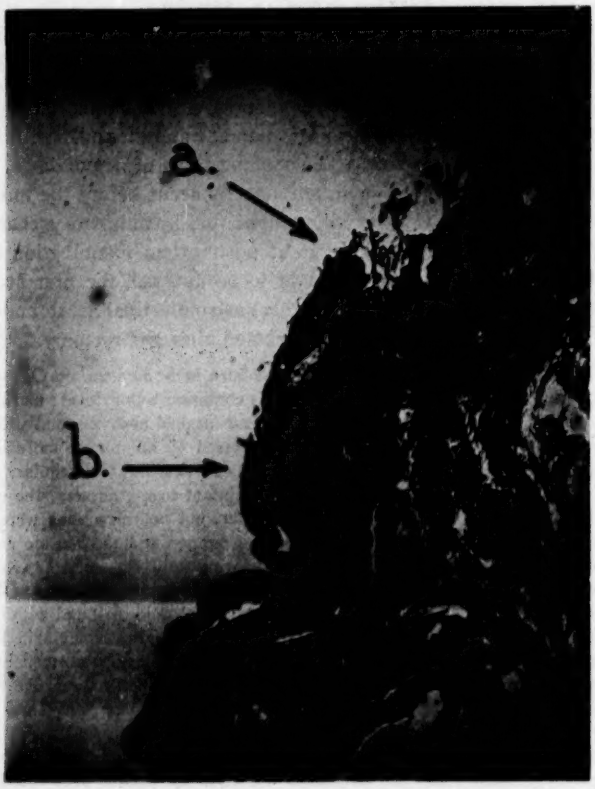

Fig. 1 (Case 1).—Photomicrograph. The lower margin of the esophageal ulcer ends at *a*. Esophageal and gastric mucosa join at *b*.

agitis and esophageal ulcer fall into three groups: (1) acute ulceration associated with asphyxia or intracranial trauma*; (2) peptic or reflux esophagitis due to inadequacy of the mechanism of the cardia,† and (3) infectious esophagitis, most commonly caused by Candida albicans in debilitated infants,‡ but rarely caused by diphtheria, tuberculosis, or syphilis.

* References 1 and 2.

† References 3 and 4.

‡ References 5 and 6.

The first description of an esophageal ulcer causing hemorrhage and death of a newborn infant is attributed to Henoch (1881).[7] When death occurred on the fifth postnatal day, an ulcer 4 cm. long was found encircling the esophagus just above the cardia. Monro's case,[8] reported in 1896, was quite similar to ours. Diamantopoulos [7] was able to find 13 cases in the literature by 1926 and added one of his own. Subsequent case reports have appeared § but are not numerous. Gruenwald and Marsh [2] found esophagitis in 52 of 310 infants examined post mortem. It was the probable cause of death in three cases in which perforation occurred, but not in three cases with hemorrhage. White,[11] reviewing 1,437 autopsies at the Children's Hospital in Boston, found 83 esophageal ulcers, but only 62 gastric and duodenal ulcers. He states that ulcers in children are small, acute, multiple, and always associated with other serious conditions. Cushing [12] reported three adult patients with ulcer of the esophagus with perforation (two with hemorrhage) and suggested a neurogenic mechanism for their production. Penner and Bernheim [1] believe that asphyxia and shock are the important causative factors; decreased blood volume with arteriolar contraction and anoxemia is followed by increased permeability of the capillary endothelium with transudation of plasma, and later cellular elements, into the surrounding tissues. The process is said to result in necrosis of the tissue in the area supplied by the affected blood vessels. Gruenwald and Marsh [2] endorse the view that hematemesis or melena in an infant is often indicative of an acute ulcerative lesion. Immediate investigation must be made to detect asphyxia, determine its cause, and remove it. Mosher [9] ascribed to infection the periesophageal and intramuscular hemorrhages of the terminal esophagus in 20 infants who died within a few days after birth. Asphyxia was present in most of his patients, however, and none died from gastrointestinal hemorrhage.

The pathogenesis of the acute esophagitis which follows asphyxia and shock has not been clearly established. It is probable that "peptic digestion" is less significant in this group of cases than are the vascular changes, leading to local tissue necrosis. The caffeine sodium benzoate given our patient shortly after birth might well have led to an increased secretion of free HCl and thus been an important aggravating factor in the production of the ulcer. A review of tissue sections from recent autopsies on 65 newborn infants revealed evidence of esophagitis in three cases, none associated with massive gastrointestinal hemorrhage. It is probable that more cases would have been found if a special search for the lesion had been made at the time the autopsy was performed.

CASE 2.—This 4-week-old white baby boy was admitted to Brooke Army Hospital Aug. 26, 1952, because of vomiting, fever, and dehydration. He had been well until four days previously, when it was noted that he coughed whenever fed and vomited two or three times during the day. The next day he vomited with each feeding and regurgitated food through his nose. Two days prior to admission, he began spitting up all food both during and between feedings, and on that day he had one normal bowel movement.

Past History.—The pregnancy and delivery were normal and uncomplicated. The child was thought to have a lot of mucus for 36 hours after birth. He was started on feedings of evaporated milk formula and had been doing well, except that he would not take more than 60 ml. at one feeding. He had gained more than 1 lb. (454 gm.) over his birth weight.

Physical Examination.—Height 20½ in. (52 cm.); weight 8¾ lb. (3,970 gm.). He frequently regurgitated small amounts of water and mucus; dehydration was marked. When given

§ References 9 and 10.

water from a bottle, he immediately regurgitated it both through the nose and the mouth. Examination of the ears, nose, and throat revealed no abnormality. Breath sounds were harsh, and gurgling sounds seemed to be transmitted from the pharynx. The lung fields were clear, heart sounds were normal, the abdomen was soft, no masses were palpable, and the remainder of the physical examination was within normal limits.

Laboratory Examination.—WBC 16,150 with 54% neutrophiles, 43% lymphocytes, 2% monocytes; RBC 4,000,000; hemoglobin 12.0 gm. per 100 ml.; BUN 23.4 mg. per 100 ml.; chlorides 97.5 mEq. per liter; CO_2 50 vol. %. Roentgen-ray study with a barium swallow revealed a moderately dilated esophagus with tertiary contractions prominent in the mid-portion. The lower third of the esophagus narrowed smoothly and appeared to end at a point just above the diaphragm. No barium passed into the stomach. Reverse peristalsis occurred with some regurgitation and some spillage into the larynx. The findings were those of a spasm at the lower end of the esophagus. No disease was demonstrable in the lung fields or the mediastinum.

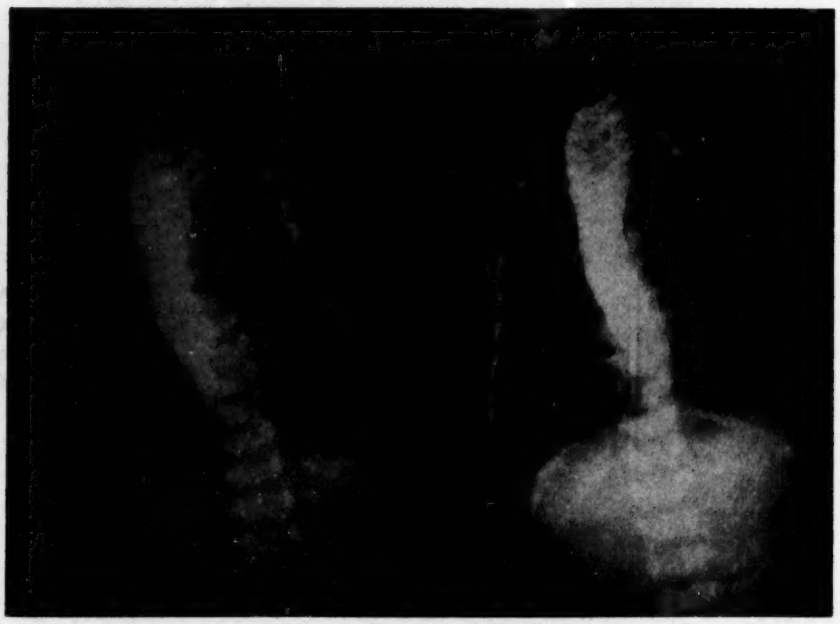

Fig. 2 (Case 2).—Initial roentgenogram showing complete obstruction of the esophagus a short distance above the diaphragm. Although there is a definite outpouching on the right wall which might represent an ulcer crater, it is thought that this defect is due to functional spasm of the esophagus, secondary to the reflux esophagitis which has produced the obstruction.

Hospital Course.—The subcutaneous administration of fluids improved the state of hydration. He continued to regurgitate all water taken orally. On Aug. 30, a gastrostomy was made. The following day, all feedings were given through the gastric tube and were retained well from the start. He left the hospital Sept. 12, 1952. The weight continued to increase; water by mouth was retained Sept. 17, iodized oil U. S. P. (Lipiodol) by mouth passed freely into the stomach Sept. 24. On Oct. 1 he had been eating well by mouth, and the gastrostomy tube was removed. By Oct. 30 he seemed to be completely well, and solid feedings were begun.

He was recalled for follow-up studies at 7 months of age. His height, weight, and physical and mental development were normal. There was no difficulty with feedings. Roentgenoscopic studies showed normal passage of the barium through the esophagus and into the stomach; strong pressure on the abdomen with the infant in the 45-degree Trendelenburg position caused

reflux of the barium only about 2 cm. above the diaphragm. Spot films made at this time disclosed a thin radiolucent shadow crossing the barium-filled esophagus about 2 cm. above the diaphragm. Longitudinal linear markings were present below this area and passed down to the stomach.

Comment.—Esophagitis, esophageal erosions, and esophageal ulceration are but different degrees of severity of the same pathologic process. The chronic peptic ulcer of the esophagus described by Albers, Rokitansky, and Tileston is a large penetrating lesion, just above the cardiac sphincter, with the histologic characteristics of a gastric ulcer and surrounded by "ectopic" gastric mucosa. Barrett [13] has pointed out that this lesion is probably a gastric ulcer in a hiatus hernia. The peptic ulcer of the esophagus described by Jackson,[14] Allison,[15] and others is quite different; the mucosa of the distal esophagus is fiery red, edematous, and congested. It bleeds easily. Erosions occur, followed by ulceration, gray-white or yellowish patches of superficial necrotic membrane surrounded by a well-demarcated border of fiery-red edematous tissue. If swabbed, a bleeding raw surface is revealed. Inflammation may involve deeper layers and lead in time to fibrous stricture and shortening of the esophagus. This lesion is thought to be due to incompetency of the mechanism of the cardia and reflux of acid gastric juice into the distal esophagus. Several anatomical and physiological mechanisms combine to form the "mechanism of the cardia," preventing reflux of gastric contents into the distal esophagus from the stomach: 1. The cardiac sphincter is a physiologic, if not an anatomic entity. 2. The tangential entrance of the esophagus into the stomach permits a fold of gastric mucosa to form a valve-like flap preventing regurgitation. 3. That portion of the esophagus which is in the abdomen is angulated. 4. There is a constrictor action of the diaphragm at the hiatus, particularly in inspiration. The insufficiency of the cardia may be due to a congenital hiatus hernia, or, much less commonly, a congenitally short esophagus due to failure of the complete descent of the stomach by the time the diaphragm fuses to form the esophageal hiatus. A very important factor in infants is the recumbent position, which encourages reflux into the esophagus.[16] Reflux of acid gastric juice into the esophagus is not the only factor in the production of esophagitis, however. Patients with cardioesophageal relaxation have frequent vomiting and reflux, but esophagitis does not develop.[4] It is probably necessary for the material to be trapped in the distal esophagus from time to time to produce the lesion. Stricture formation may prevent regurgitation and thus permit healing of the esophagitis proximal to the stricture.

A definitive diagnosis of esophageal ulcer cannot be made without visualization of the lesion endoscopically or at surgery or autopsy. The lesions are seldom deep enough to be apparent on the roentgenogram, but functional spasm with outpouchings and constrictions of the lumen are regularly present and often misinterpreted as ulcer craters (Fig. 1). The demonstration of longitudinal striations above the level of the diaphragm is good evidence of herniation of a portion of the stomach into the thorax.

We feel that the patient in Case 2 has a small congenital hiatus hernia of the sliding type. This caused some difficulty in feeding from birth, and the anatomical defect permitted the development of "reflux esophagitis" with inflammatory stricture which proceeded to complete obstruction. Gastrostomy allowed his nutrition to be maintained, and the obstruction prevented further reflux of gastric contents into the esophagus. Healing has now occurred, and the roentgen-ray examination at the age

of 7 months demonstrates a small hiatus hernia of the sliding type. Esophagoscopy should be performed when he is a little older, and if esophagitis is still present consideration should be given to surgical repair of the hernia. Medical measures were sufficient in 75% of Carré's 112 patients,[17] and only 10% required surgical repair.

SUMMARY

Hemorrhage from the gastrointestinal tract in newborn infants is a usually lethal complication of "stress ulcer." Because of its frequent association with asphyxia, hemorrhage may call attention to previously undetected interference with respiration.

A case is reported of fatal gastrointestinal hemorrhage from a small linear esophageal ulcer just above the gastric cardia of a newborn premature infant. Asphyxia and probably intracranial trauma are thought to have been important causal factors in the production of this ulceration.

Reflux esophagitis may occur in very young infants if a sliding type hiatus hernia is present. The mechanism is the same as in the adult. Such a case is reported in a 4-week-old infant. Complete obstruction of the esophagus occurred, but relented three weeks after gastrostomy was performed. The obstruction is thought to have been the result of inflammatory edema and stricture.

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Case Reports

IDIOPATHIC CALCINOSIS UNIVERSALIS

Report of a Case in a Child Treated with Corticotropin (ACTH) and Cortisone

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LOS ANGELES

CALCINOSIS is the term applied to the abnormal deposition of calcium salts into the skin, subcutaneous tissues, muscles, and tendons. The disease is usually divided into two clinical types, depending upon whether the deposit is localized (calcinosis circumscripta) or generalized (calcinosis universalis). The latter type is the severer of the two and is the variety usually observed in children and young adults. The condition is uncommon, and only sporadic reports have appeared in the pediatric literature.

The cause and pathogenesis are obscure; however, certain workers [1] believe that the condition is related to scleroderma and dermatomyositis. Because of the reported efficacy of corticotropin (ACTH) and cortisone in certain of the collagen diseases and in view of the lack of a specific treatment for calcinosis, we elected to administer these hormones in this case.

REPORT OF CASE

This 11-year-old girl was admitted Aug. 31, 1951, with a chief complaint of "lumps" on her leg. Approximately one year before admission, the patient fell aind bruised her right leg. The area was treated with local antiseptics, and healing was uneventful. Approximately four months later, subcutaneous nodules developed in the area previously injured. After their initial appearance the nodules increased in size very slowly and were not painful.

In July, 1951, the nodules became tender and pustular. They were incised and drained. The incisions remained open and an "exudate" which was first yellow and later white continued to drain from the sinuses on the leg. Examination of the drainage showed calcific material. The patient was admitted to our service for a diagnostic work-up.

Family history was noncontributory, except that one sister had asthma and allergic dermatitis (eczema). She received the usual immunizations and vaccination. Pregnancy and delivery were uneventful. Her birth weight was 8 lb. 9 oz. (3,880 gm.). She was born with an area of depigmentation over the right forehead extending into the hair. She had German measles, chicken pox, pertussis, and otitis media during the first and second years of life. Tonsillectomy and adenoidectomy were done at 5 years. Her early development was normal. At the time of admission she was in the sixth grade and was characterized as a good student.

In view of the history of allergy in a sibling, intracutaneous tests to common environmental inhalants and foods were done. The results of these were all negative except for a positive reaction to house-dust extract.

Physical examination on admission revealed a moderately well-developed tall thin 11-year-old Negro girl (Fig. 1). She was cooperative, alert, and in no distress. Physical examination of the head, neck, chest, and abdomen disclosed essentially normal findings, with blood pressure 110/80, pulse 92, and temperature 99.8 F.

From the Department of Pediatrics, Howard University College of Medicine, and the Pediatric Service of Freedman's Hospital.

There was a pustular area on the left elbow that the patient stated had been present for about one month. The surrounding tissue exhibited hyperemia, induration, and crusting. On pressure, a light yellow substance exuded. The lesion was not painful. There were several nodules observed on the middle third of the anterior surface of the right leg (Fig. 2). There was evidence of inflammation at the site of at least two of the nodules which presented draining sinuses. Several other subcutaneous nodules showed no signs of inflammation. The nodules were not painful to palpation. Similar subcutaneous excrescences could be palpated beneath the skin over the left patella.

Fig. 1.—Photograph of patient whose habitus was of the asthenic type. The bandage on the leg indicates the approximate location of several subcutaneous nodules and perforating lesions.

Laboratory Findings.—On admission the hemogram showed the following: red blood cell count 3,340,000, white blood cell count 4,650, hemoglobin 11.1 gm. per 100 cc., hematocrit reading 37%, and erythrocyte sedimentation rate 17 mm. in one hour. The initial total eosinophile count was 750 cells per cubic millimeter, however, there was a prompt and sustained fall to less than 150 cells during hormone administration. The blood chemistry was as follows: nonprotein nitrogen 26 mg., glucose 76 mg., serum calcium 10.3 mg., serum phosphorus 7.1 mg., albumin 3.6 gm., and globulin 2.86 gm. per 100 cc., serum chloride 99 mEq. per liter, alkaline

phosphatase 10.4 Bodansky units. Initial serum sodium and potassium values were normal. Blood Kahn reaction was negative. Results of urinalysis were normal. The basal metabolic rate was + 6%, and findings on the electrocardiogram were normal.

Cultures from lesions on the leg and elbow grew a hemolytic Staphylococcus aureus, mannitol-positive with antibiotic sensitivity as follows: chlortetracycline (Aureomycin) 1.5 γ, oxytetracycline (Terramycin) 3.1 γ, and penicillin 25 units per cubic centimeter. Nose and throat cultures revealed alpha- and beta-hemolytic streptococci, nonhemolytic Staph. aureus and Hemophilus hemolyticus. A smear made from the cutaneous lesion on the elbow showed many polymorphonuclear leucocytes and lymphocytes, but no eosinophiles.

Fig. 2.—Photograph of middle third of right leg showing subcutaneous nodules, two of which have perforated. The draining cutaneous sinuses exuded calcareous material. They healed on hormone therapy.

Biopsy of a typical lesion involving skin and subcutaneous tissue disclosed a calcium content of 5.5%, whereas the calcium content of the normal skin on which a biopsy was done was negligible (trace only). (Normal skin contains approximately 0.014% calcium.[2])

During the period of hospitalization (Sept. 18 to Oct. 5, 1951) the patient received 40 mg. of corticotropin daily by intramuscular administration. At the end of this treatment period there was no appreciable change in skin or subcutaneous tissues on inspection or by x-ray examination.

There were no significant untoward reactions related to corticotropin administration. At the time of discharge from the hospital she was started on cortisone therapy because of the ease of oral administration of this at home. From Oct. 5, 1951, to Feb. 5, 1952, she received 25 mg. daily. During this period all discharging sinuses healed, and the child's disposition improved significantly. On Jan. 22, 1952, a tender but noninflammatory nodular swelling appeared on the medial aspect of the left knee. On Feb. 5, 1952, the dose of cortisone was increased to 50 mg.

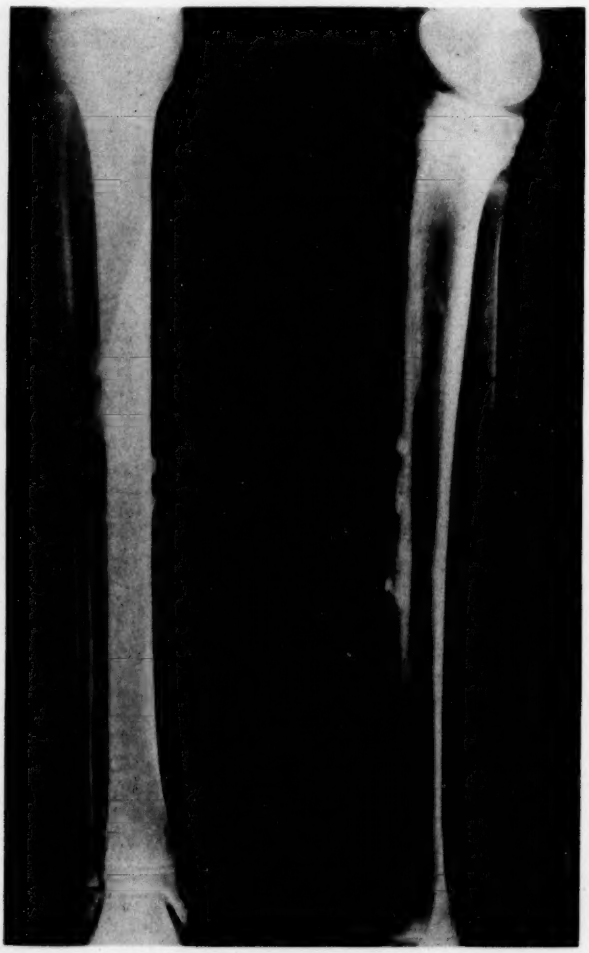

Fig. 3.—X-ray of right leg before hormone therapy. There are numerous calcific deposits in the soft tissues, with greatest concentration in the middle third.

daily (oral) because x-rays showed additional calcification about the left knee. She remained on this dosage until April 15, 1952, when it was discontinued for an interval.

At this time all open lesions were healed, and there was no drainage anywhere. Small subcutaneous excrescences could be palpated on the medial aspect of the left knee and over the anterior surface of the right leg. These areas, however, showed no evidence of tenderness or acute inflammation. The patient was enjoying good general health and offered no complaints.

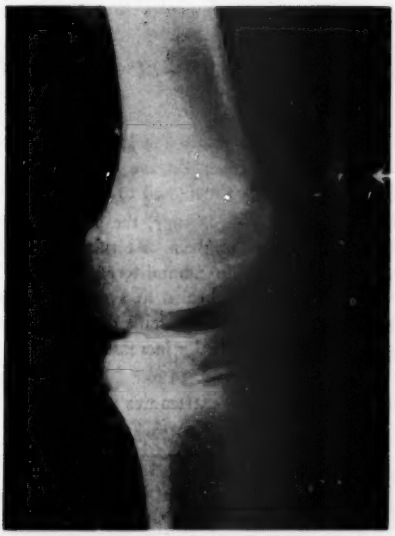

Fig. 4.—X-ray of left knee before hormone therapy. Note calcific deposit in prepatellar area.

Fig. 5.—X-rays of right leg and knee made March 7, 1953, after hormone therapy. Film reveals increase in calcific deposits about the knee and no essential improvement in the status of similar deposits in the leg.

In mid-July, 1952, the subcutaneous nodules previously described in the region of the left knee began to increase in size, hence cortisone therapy was resumed (25 mg. administered orally daily). In December, 1952, the nodules became larger, more superficial, and somewhat tender. The dose of cortisone was therefore increased to 50 mg. daily.

In early January, 1953, despite hormone therapy, the status of the nodules was such as to warrant incision and drainage of several of them. At this time the therapeutic regimen was modified to include cortisone and penicillin administered internally and bacitracin ointment topically. On this regimen the open lesions healed in a period of three to four weeks. However, as shown in Figures 5 and 6, the soft-tissue calcific deposits remained essentially unchanged in the legs, whereas those about the knee actually increased in size.

X-ray Findings.—Initial x-rays, taken Aug. 14 and 16, 1951, of the skull, vertebral column, chest, abdomen, pelvis, and upper and lower extremities revealed normal findings except for the

Fig. 6.—Anteroposterior and lateral views of left knee after hormone therapy. An actual increase in the amount of calcareous material has occurred. (Compare with the pretreatment film in Figure 4.)

following: Anteroposterior and lateral films of the right leg (Fig. 3) disclosed numerous calcified masses in the soft tissues, with greatest concentration in the middle third of the leg and near the knee joint. In some areas the smaller deposits coalesced to form larger conglomerate masses. The tibia and fibula were not involved and presented normal architecture. Films of the left lower extremity (Fig. 4) revealed calcific deposits concentrated in the prepatellar soft tissues. The bones of the knee joint were normal in appearance.

Follow-up films of the lower extremities taken on Jan. 22 and Feb. 5, 1952, showed no significant change from the previous examination except that there was now an increase in calcific deposits about the left patella and the femoral condyles.

Subsequent films of the legs taken April 22, 1952, showed only a slight decrease in soft-tissue calcific deposits at the knees and at the middle third of the right leg.

X-rays of the legs made on May 31, 1952, revealed no essential change from the previous examination.

Final films of the lower extremities (Figs. 5 and 6) made March 7, 1953, showed an increase in calcium deposits in the prepatellar soft tissue and a persistence of similar deposits in the soft tissue of the middle third of the leg.

ADDENDUM.—When last observed, on Sept. 14, 1953, this child presented several noninflammatory painless excrescences about the knees and the middle third of the anterior surface of the right leg. She was still receiving cortisone daily in a dose of 50 mg. administered orally.

COMMENT

Many theories of the cause of calcinosis have been advanced, but none completely accepted.[3] It is not clear why the condition occurs more frequently in women.[4]

The symptomatology usually depends on the location of the calcium deposits. Provided no infection occurs, the patient is incapacitated only by the contraction, limitation of motion, and pain due to the pressure of the calcified masses in the skin, muscles, or joints. The subcutaneous nodules created by the calcific deposits often ulcerate and become secondarily infected, as occurred in this case. A chalky gritty material of diagnostic significance may be recovered from draining sinuses. Diagnosis can be confirmed by x-ray of the part and biopsy of the lesion.

Pathologically, the nodules reveal the presence of granular particles of calcium salts embedded in the interstices of the fibrous connective tissue or subcutaneous fatty tissue. Inflammation with foreign-body giant-cell infiltration is usually present. Chemical analysis of the discharge from the lesion usually shows increased concentration of calcium phosphate and carbonate salts over the normal levels.

The prognosis is variable. The disease is characterized by chronicity, remissions, and exacerbations. There is no specific therapy. Varying degrees of success have been reported for parathyroid extracts, acid salts, ketogenic diet, x-ray irradiation, physical therapy, and surgical procedures such as parathyroidectomy.[5]

Briggs and Illingworth[6] reported the cases of two children treated with hormones. In one of the cases (4-year-old girl) in which the calcinosis followed dermatomyositis, the disease appeared to respond dramatically to corticotropin therapy, with almost all calcium deposits disappearing during the course of treatment. In the second case (13-year-old girl) which was of many years' duration and unrelated to dermatomyositis, the disease did not respond to either corticotropin or cortisone therapy.

The case presented here exhibits the typical clinical and x-ray features of calcinosis universalis.[7] Under hormone therapy the perforating lesions (cutaneous sinuses) healed, and the patient's disposition improved. However, new lesions subcutaneous nodules) developed during hormone administration, and old ones increased in size. Moreover, except for minor remissions the soft tissue calcareous deposits, which were followed by x-ray over an 18-month period, failed to resolve under hormone therapy.

SUMMARY

Calcinosis universalis is uncommon in childhood. The disease is characterized by remissions, exacerbations, and chronicity. Heretofore, treatment has been unpredictable and generally unsatisfactory.

Because of the reported beneficial results of cortisone therapy in other collagen diseases, this hormone was administered to our patient for a period of more than

one year. Serial x-rays were made over a period of 18 months. According to the x-rays, there was no significant improvement in the status of the soft-tissue calcium deposits during therapy.

Dr. Lloyd H. Newman, Department of Biochemistry, performed the chemical analyses on biopsy material from the skin and subcutaneous tissues for calcium content. The cortisone (Cortone) was furnished by the Medical Division of Merck & Company, Inc., Rahway, N. J. Corticotropin (Acthar) was supplied by the Armour Laboratories, Chicago.

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Society Transactions

CHICAGO PEDIATRIC SOCIETY

George F. Munns, M.D., *President*
Regular Meeting, March 17, 1953

Symposium: The Care of the Handicapped Child

Medical Aspects. Dr. Abraham Levinson.

The care of the handicapped reflects the advances of medicine and society in the last few centuries. From the time when those who were deformed or retarded were thrown into the river or abandoned until the present day, the care of the handicapped represents the barometer of civilization. In our present handling of the problem much of the advance has been due to medical knowledge, and the handicapped child particularly has become the concern of the physician.

The physician must learn to recognize the child as a whole, and the importance of complete physical examination cannot be overemphasized. In this age of specialization, the orthopedist examines the hip, the ophthalmologist looks at the eyes, and so on. Pediatricians must be specialists on the whole child and recognize a handicap even when the cause is obscure. Any handicap should be recognized early. Years ago we saw children in the chronic stage of many diseases which today we recognize and treat early.

Nutrition is important. One of our great problems is anemia, and the pediatrician should be on the alert for this. Gingivitis is an important complication in the handicapped child. Many children have scoliosis, not because they have spinal trouble, but because they are bedfast for some other condition. Poliomyelitis is a pediatric problem, with the orthopedist as a consultant. In psychologic and psychiatric problems, the pediatrician should play a major role. We should not treat the disease, we should treat the child as a whole.

Emotional Aspects. Dr. Julius B. Richmond.

Whether we are concerned with the so-called normal or the handicapped child, our efforts are in the direction of helping each to become a happy, effective, and productive citizen who can contribute to, as well as take from, the community in which he lives. How well he attains these objectives will depend to a considerable extent upon the emotional interrelationships he has within his family and his community. The handicapped child and his family have problems, many of which are unique in relationship to the specific handicap. In our professional roles we must help in the resolution of these problems. We cannot solve them entirely, for the real solutions come from the patient and family.

The provision of really adequate physical or medical care has fundamental psychologic significance. Unless adequate care is provided, both child and family may be expected to feel insecure. Good physical care should be emphasized, lest in the provision of psychologic service we lose sight of the forest for the trees.

The child with a handicap faces problems relating to development of a feeling of being wanted or accepted. Unless the parents are able to manifest warmth toward the child, a feeling of rejection and insecurity may develop. To these the child must develop defences. These may center about withdrawal (such as inability to discuss his problem or attempted concealment of it) or hostility which may be manifested by undue aggressiveness. These forms of behavior are often difficult to cope with, and it becomes important that they be dealt with in an understanding rather than a punitive way.

Parents of all handicapped children experience to some degree feelings of guilt about the handicap—whether it be acquired or congenital. Realistic assurance that the parent has no direct responsibility is not sufficient to allay these feelings. The lack of realistic basis for these feelings is often revealed when parents who obviously have the intellectual capacity to know better will

ask about whether animal impressions during pregnancy may have produced a congenital anomaly, or whether some physical punishment had anything to do with an acquired injury. The physician should provide an objective and sympathetic audience, together with confidence in terms of professional ability. In this way the parent has an opportunity to relieve himself of some of these hostile feelings and at the same time to share the anxiety with a person of status in the community. This may make the physician a very close friend of the family effort in the face of hopeless handicaps for which he realistically can do but little. We have all seen examples of devotion of families to physicians who care for children and with such disorders as progressive muscular dystrophy, for example, when the physician can do little for the child, yet the family's gratitude is out of all proportion to his realistic contribution. His sharing of anxiety with the family offers them a safety valve which provides great relief and comfort.

In every program for handicapped children the success of physiologic management depends upon the development of an individual program for each child. This means a convergence of medical, physiologic, social, and educational services in the direction of constructive planning. No one of these services provides all the help. Rather, it is the unique contribution of each, combined into a total program which will provide most effectively for all handicapped children. The effectiveness of this integration of services is to a large extent a measure of the integration and maturity of a community. How well the community accepts all citizens, including its handicapped, and plans effectively for them is an index of the level of our civilization.

Special Education. MARY E. COURTNEY.

At the present time there are approximately 23,000 of the more than 400,000 children enrolled in the Chicago Public Schools for whom special learning experiences and situations must be supplied. About 1,400 of these, plus some 2,000 enrolled yearly in the hospital instruction and home instruction programs, are physically handicapped youngsters. They are housed in four special schools. Restrooms, clinics and treatment centers, ramps, wheelchairs, walkers, and all manner of specially devised equipment are as essential as classrooms and laboratories. The furniture is moveable and adjustable and is supplemented by skillful contrivances to meet the particular needs of the individual student.

Children are transported by school buses under the supervision of qualified attendants, who also care for those who need help throughout the daily routine. A staff of qualified physical therapists administer physical therapy, hydrotherapy, and recreational therapy, as prescribed by the medical authority on whose recommendation the student is enrolled. No child enters one of these schools except on the basis of a medical form which carries a specific diagnostic statement and prescription of treatment by physician, hospital, or clinic.

For those who cannot attend school, the hospital and home instruction programs provide the services of qualified public school teachers. These expanding programs are the latest development in the schools that serve the physically handicapped.

Some 1,100 boys and girls, partially sighted and totally blind, profoundly hard-of-hearing and deaf, are cared for in special classes and centers. Qualified teachers instruct them in the special techniques employed by the blind and in speech and language development and speech reading for the deaf. They are gradually integrated into all school activities and enjoy normal learning experiences with other children.

The dearth of teachers finds acute expression in these highly specialized fields. Hence, a recruitment campaign is a constant and significant factor in the whole program.

Social Agencies. DR. EDWARD PRESS.

One of the major public agencies that helps patients of pediatricians is the Chicago Pediatric Society, through its individual members who contribute their time in outpatient clinics and wards, and in the care of indigent patients. There are many public agencies which can be of aid in such a program, through public health nurses, teachers, social workers, and provision of artificial limbs, braces, hearing aids, and other items. By learning of these agencies and using their facilities, pediatricians may get needed services for many of their patients who would otherwise do without or with less than is available.

The Chicago Association of Commerce and Industry lists 226 civic, health, and welfare agencies that they endorse. In addition, there are many that are not endorsed and also the official tax-supported agencies. For the many voluntary agencies operating in the community, the Community Referral Service should be contacted.

A list of the major official or governmental public agencies was given and a brief description of the services of the Division of Services for Crippled Children of the University of Illinois, together with illustrations of sample cases.

State Institutions. DR. WILLIAM W. FOX.

The Department of Public Welfare operates the Lincoln State School and Colony, located at Lincoln, and the Dixon State Hospital, at Dixon. These two facilities care for all types of mentally handicapped in all age groups. Programs are designed for the noneducable as well as for the educable. It is estimated that there are approximately 80,000 retarded persons in the state, and at this time the institutions are greatly overcrowded.

Prior to 1940, the request for admissions of children under the age of 6 was not very noticeable. However, since then the trend has been to recommend institutionalization at an early age for such children as the Mongoloid types, hydrocephalics, microcephalics, and other gross physical anomalies associated with retardation. There are waiting lists at both institutions, totalling 204. A program of expansion has been under way since the end of the war.

The question arises as to when to institutionalize an infant or child. Naturally, this depends on many factors: whether or not the parents are emotionally stable to accept the retarded child; whether or not the home is adequate, and whether or not the child is antisocial and fails to make proper adjustments in the community because of lack of proper supervision.

Many times the question arises as to whether a newborn Mongoloid child should be taken home. The general hospital does not want to keep the infant, since it needs the cribs for others. The cost of retaining a child of this type either in the hospital or a private nursing home is a tremendous financial burden on the parents. It is believed that this type of child can be easily cared for at home, inasmuch as the care is not different than that for other infants. A criteria for emergency admission to one of the institutions has been developed based on the diagnosis, the kind of care the child is receiving, the kind of care he requires, the attitude of the parents toward the infant, the health of the parents, and the financial status.

A visit to either institution will give a clearer picture of the problem, and of what is being done through the Department of Public Welfare.

The National Society for Crippled Children and Adults. EVELYN JACOBS.

George F. Munns, M.D., *President*
Regular Meeting, April 21, 1953

Borden Award for Pediatric Residents

Adrenal Syndromes in Childhood and Adolescence. DR. DOROTHY LYNCH.

Eleven cases of genital syndromes in children and adolescents are described and illustrated. The problems encountered in definitive diagnosis and the diagnostic procedures employed are discussed.

Emphasis is placed on the early differentiation of sex in relation to the parental attitudes and emotional growth of the child.

Recent hormonal therapy is reviewed with reference to the author's experience with cortisone therapy in three cases of congenital adrenal hyperplasia.

Anatomic Variations in Tetralogy of Fallot. DR. THOMAS BAFFES and DR. FRANK JOHNSON.

The 42 specimens of this disease available in the heart collection of the Children's Memorial Hospital were examined. It was found that 12 of these hearts had either complete or partial pulmonary atresia and therefore could not have had intracardiac resection. Only four of the 12

had remnants of the pulmonary artery large enough to permit a shunt operation. The remaining 30 hearts were controversial, inasmuch as they could be treated either by intracardiac surgery or by systemic-pulmonary shunts.

Since no instance of pure valvular stenosis without infundibular deformity was found, the 30 controversial cases were categorized into five main groups, depending upon the type of infundibular stenosis noted. It was found that 37% could have been treated by intracardiac surgery, while 63% would have had to be treated by the shunt operation.

These findings differed markedly from those of Brock and Glover and co-workers, who found that in a large proportion of their tetralogies there was pulmonary valvular stenosis without infundibular deformity and also noted that the infundibular deformities present were such that intracardiac resection was possible in many instances. It was noted, however, that our series consisted primarily of cases of patients less than 3 years of age, while that of Brock and Glover and co-workers contained predominantly patients above that age.

It is apparent that our cases must have been of severer tetralogies, in which the patients usually do not reach adolescence or adulthood before operation must be attempted. In view of the small size of the heart and the severity of the infundibular deformity in these younger hearts, the shunt operation would seem to be the procedure of choice. In the older patients with tetralogies, with valvular stenosis, or with less severe infundibular deformity, intracardiac operation may be indicated.

One other factor to be considered is the severity of overriding of the aorta. The removal of the infundibular obstruction should permit flow of blood into the pulmonary artery and therefore decrease the severity of the right-to-left shunt via the interventricular septal defect. However, in our series the overriding of the aorta was usually so severe that some right-to-left shunt would probably have persisted despite removal of the infundibular obstruction. The size of the pulmonary artery had little influence upon the hypothetical choice of operation. Wherever there was a very small pulmonary artery or severely stenotic pulmonary valve, it was associated with infundibular stenosis so severe that a shunt operation was preferred.

We feel that the shunt operation has a wide applicability in this condition, particularly at the younger ages, where the infundibular deformity is severe, the overriding of the aorta is marked, and the pulmonary valvular stenosis without infundibular deformity is rare.

Subcutaneous Administration of Oxytetracycline (Terramycin). Dr. R. S. Mendelsohn.

Oxytetracycline (Terramycin), with its almost complete absence of serious toxic effects and the low incidence of distressing side reactions, has become a drug of choice in many infections in pediatric practice. In January, 1952, studies were undertaken to determine whether this antibiotic could be given by parenteral routes other than the intravenous.

Forty infants were treated with a buffered preparation of oxytetracycline hydrochloride given subcutaneously. The infants had a variety of diseases. Their ages ranged from prematurely born infants 6 hours of age to infants 1 year old. All doses were given in a concentration of 1 mg. per cubic centimeter, except in one case. The fluids used for dissolving the oxytetracycline were normal saline, 5% dextrose in normal saline, dextrose in distilled water, or distilled water. A dose of 5 mg. per pound (0.5 kg.) was given every four hours.

There were no untoward effects, either local or general. Clinical results and studies on the absorption showed that this is an effective route of administration. It is practical, easy, apparently free from untoward effects, and effective.

This route of administration further enhances the usefulness of this valuable wide-spectrum antibiotic, and its application is of practical value in pediatric practice.

Correspondence

FOLLOW-UP OF A BREAST AND BOTTLE SHY INFANT

To the Editor:—It was stated in "A Breast and Bottle Shy Infant at 21 Years of Age" (Feldstein, G. J.: Am. J. Dis. Child. **77**:374 [March] 1949), a follow-up report of "A Breast and Bottle Shy Infant, Nursing Only When Asleep" (Feldstein, G. J.: Am. J. Dis. Child. **35**:103 [Jan.] 1928), that if she marries and has any children, it will be of interest to note whether this trait appears in any of her offspring. She has since married and was delivered of a full-term normal infant July 6, 1952. She had a normal pregnancy, delivery, and puerperium, the only problem being one of weight control; she gained 39 lb. (18 kg.) during her pregnancy. The infant was 7 lb. 14 oz. (3,570 gm.) and 21 in. (53.3 cm.) at birth and 20 lb. (9,070 gm.) at 15 months. The bottle was well taken, but there was difficulty in getting him to take food from a spoon; he refuses the junior canned baby foods. He is allergic to bananas and sweet potatoes, which invariably produce diarrhea in him. While this infant shows some evidence of negativism and minor feeding difficulties, these are not much more marked than are frequently noted in other infants and children at certain periods of their development. The mother's refusal to nurse him makes it impossible to give an opinion as to breast shyness, but the absence of bottle shyness and nursing only when asleep shows that he has not inherited the very unusual traits reported in his mother in her infancy.

George J. Feldstein, M.D.
Pittsburgh

Obituaries

EDWIN E. GRAHAM, M.D.

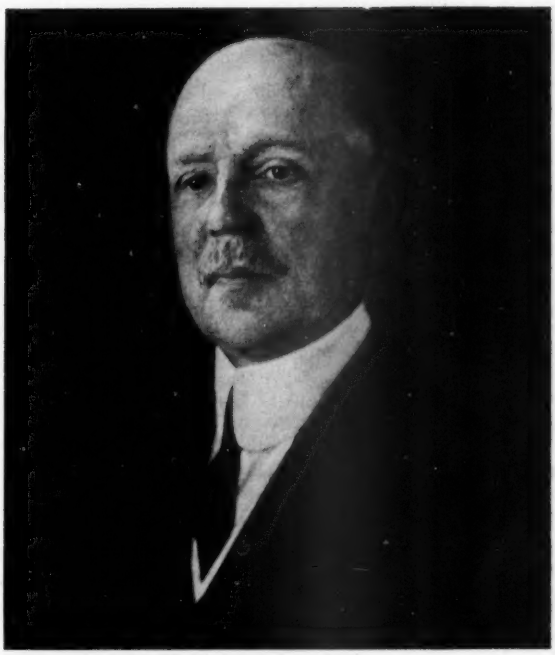
1864-1953

Dr. Edwin E. Graham, Emeritus Professor of Pediatrics of Jefferson Medical College, died June 10, 1953, while visiting a daughter, Mrs. Edwin A. Bacon, in Madison, Va.

Dr. Graham, born Feb. 28, 1864, in Philadelphia, received his B.A. from Harvard, in 1884, and his M.D. from Jefferson, in 1887. Following a year's internship, he studied in Göttingen, Berlin, and Munich for a year. On Jan. 2, 1893, Dr. Graham married Lorraine Goodrich. In 1904, he was appointed to the first Professorship of Pediatrics established at Jefferson Medical College of Philadelphia. Elected to the American Pediatric Society, in 1897, he was President of that society in 1919. He was also a member of the American Medical Association, the Philadelphia Pediatric Society (past-president) and other societies, and, in 1950, was given an honorary degree of Doctor of Science by Jefferson Medical College.

Dr. Graham was the author of "Children's Diseases," a textbook for students; he published many articles on pediatric subjects and for several years was on the editorial board of the AMERICAN JOURNAL OF DISEASES OF CHILDREN. He also contributed to Hare's and to Wilson's textbooks on therapeutics.

Few, if any, of Dr. Graham's contemporaries survive him. However, reading between the lines which list his formal accomplishments, one realizes that Dr. Graham occupied for many years, as teacher, writer, and physician, a respected position among the pioneers in pediatrics in this country.



EDWIN E. GRAHAM, M.D.
1864-1953

Abstracts from Current Literature

Newborn

Pulmonary Hyaline Membranes in the Newborn. Jens Marstrander, Bull. Tulane Med. Fac. **12**:118 (May) 1953.

The problems of pulmonary hyaline membranes are discussed. Theories as to the pathogenesis are brought out, and unanswered questions are mentioned. At the present state of our knowledge all that can be done is to institute precautions to avoid any aspiration—in other words, to observe the cardinal principles of resuscitation in the newborn.

McGuigan, Evanston, Ill.

Effect on Weight Gain of the Addition of Lactobacillus Acidophilus to the Formula of Newborn Infants. Edward L. Robinson and Walter L. Thompson, J. Pediat. **41**:395 (Oct.) 1952.

Robinson and Thompson studied 800 babies who were fed formulas supplemented with folic acid and/or Lactobacillus acidophilus culture. Their gains in weight were measured. From this study they concluded that infants partially nursed, even for two or three days, showed significantly greater weight gains during the first month than infants completely bottle fed. Bottle-fed infants on formulas supplemented with L. acidophilus showed significantly larger gains in weight during the first month than did the controls. However, the addition of L. acidophilus to the formula had no effect on the gain in weight of partially breast-fed infants. They conclude that there appears to be an unknown factor or factors present in colostrum which causes increased gain in weight during the first month of life. A considerable part of this increase can be obtained by supplementing the formula of the completely bottle-fed infant with L. acidophilus for the first few days of life.

Macdonald, Pittsburgh.

Staphylococcus Aureus Infections in the Newborn Infant. Felix Feldman and David Annunziata, J. Pediat. **41**:399 (Oct.) 1952.

In this epidemic in newborn infants of Staphylococcus aureus infections with protean manifestations, the treatment of choice was chlortetracycline (Aureomycin). The organisms were apparently resistant to other antibiotics.

Macdonald, Pittsburgh.

Paroxysmal Auricular Tachycardia Associated with Wolff-Parkinson-White Syndrome in a Newborn Infant. Marshall B. Kreidberg and Theodore A. Dushan, J. Pediat. **43**:92 (July) 1953.

The authors report a 48-hour-old infant hospitalized because of cyanosis and rapid respiration. In addition to the paroxysmal tachycardia and congestive failure, there were electrocardiographic evidences of the Wolff-Parkinson-White syndrome. The tachycardia responded to digitalis, and the rhythm and rate returned to normal. However, electrocardiographic evidence of the Wolff-Parkinson-White syndrome persisted.

Macdonald, Pittsburgh.

Experiments on Rectal Administration of Oxygen to Newborn Anoxic Kittens. E. Malm and H. Tallquist, Acta pædiat. **42**:313 (July) 1953.

The absorption of oxygen administered into the intestine of newborn anoxic kittens has been investigated. The results indicate that the oxygen content of the blood of markedly anoxic newborn kittens increases during rectal administration of oxygen.

From the Authors' Summary.

EFFECT OF THE HYALINE MEMBRANES ON THE LUNGS OF NEWBORN INFANTS. S. RANSTROM, Acta Pædiat. **42**:323 (July) 1953.

An analysis of 30 cases of newborn infants with hyaline membranes in the lungs has led to the conclusion that these membranes are of no significant functional importance in the vast majority of cases. In more than half of the cases death was considered to be due to pneumonia. In cases where there was no pneumonia or demonstrable extrapulmonary cause of death, the significant factor in the prevention of the gaseous exchange in the lungs was primary and/or secondary atelectasis. The atelectasis was not located only in parts of the lungs corresponding to the membranes. The clinical diagnosis of "membrane asphyxia" in the sense of "asphyxia caused by membrane formation" must be limited to cases with very abundant and wide-spread membranes causing secondary atelectasis.

FROM THE AUTHOR'S SUMMARY.

Acute Contagious Diseases

PERTUSSIS. R. V. PLATOU and J. M. HORAN, Bull. Tulane M. Fac. **12**:91 (May) 1953.

The authors present a very concise and effective discussion of the present status of our knowledge of pertussis. Diagnostic, immunologic, and therapeutic measures are covered thoroughly but briefly.

McGUIGAN, Evanston, Ill.

PASSIVE IMMUNIZATION IN RELATION TO MULTIPLE CASES OF POLIOMYELITIS IN THE HOUSEHOLD: EPIDEMIOLOGIC ASPECTS IN NEW YORK CITY, 1949-1952. MORRIS SIEGLE and MORRIS GREENBERG, New England J. Med. **249**:171 (July 30) 1953.

The epidemiology of multiple household infections occurring in New York from 1949 to 1952 was studied with particular reference to the applicability of passive immunization as a community control measure.

The percentage of households affected with more than one case of poliomyelitis in a given year varied with an annual incidence of 2.4 to 4.4

The incidence of multiple household infections was related to the number of household members under 18 years of age and to the annual attack rate in the general population.

The time interval between the onset of the initial and subsequent cases in a household followed a characteristic pattern, most of the cases occurring within the first week, about 95% within two weeks, and 99 to 100% within five weeks. The percentage of cases after the first week seemed to be somewhat higher among persons 15 to 34 than among those under 15 years of age.

The incidence of poliomyelitis in household contacts after the onset of the index case was about 40 times greater than that of the general population. It was proportionately greater at 15 to 34 than below 14 years of age. In the group from 15 to 34, the frequency of bulbar poliomyelitis and the mortality were also increased.

WHEELOCK, Denver.

PATHOLOGICAL MANIFESTATIONS IN AN INFANT AFTER MATERNAL RUBELLA IN THE SIXTEENTH WEEK OF GESTATION. C. G. TEDESCHI, M. M. HELPERN, and T. H. INGALLS, New England J. Med. **249**:439 (Sept. 10) 1953.

The authors report the case of a male infant delivered by caesarean section three weeks before term of a mother who had contracted rubella during the 16th week of pregnancy. There were cataractous changes in the periphery of the lens of the right eye and associated manifestations of acute panophthalmitis, including vascular thrombosis and striking infiltration of polymorphonuclear leucocytes of the eosinophilic type.

It is suggested that lens protein released during the embryonic period played the role of a sensitizing factor. Coexisting renal changes, mainly characterized by fibrosis and hyaline degeneration of the tufts and capsules of the glomeruli, may be interpreted as nonspecific late manifestations of the widespread attack of the virus of rubella upon the tissues of the embryo.

WHEELOCK, Denver.

LUMBAR SYMPATHECTOMY IN SEQUELAE OF POLIOMYELITIS AFFECTING THE LOWER EXTREMITIES. MARIO NOE, GUILLERMO ZIEGLER, and JORGE DEL RIO, Rev. chilena pediat. **24**:187 (June) 1953.

Lumbar sympathectomies are useful in the treatment of vascular lesions of the leg in poliomyelitis. It is an easy operation if Leriche's technique is followed. As a secondary result one can obtain less shortening of the leg on the operated side, which, together with the possibility of retarding the growth of the other leg, allows a greater total height to be obtained.

FROM THE AUTHORS' SUMMARY.

ORTHOPEDIC APPARATUS IN THE TREATMENT OF POLIOMYELITIS. LUIS SIERRA ROJAS and ALFONSO TOHEN ZAMUDIO, Bol. méd. Hosp. inf. **10**:128 (April) 1953.

Orthopedic apparatus is of prime importance in the treatment of poliomyelitis in any of its stages once the febrile syndrome has disappeared.

Orthopedic apparatus in poliomyelitis paralysis not only helps to counteract established or definitive disabilities, but is also of positive value for providing rest, to prevent bad positions, to protect weak muscles, to prevent deformities, or to correct those that are beginning, all of which help in maintaining the musculoskeletal system in the best condition to attain the greatest possible recovery.

Based on the physiopathology of the disease, we classify the apparatus in three main groups: (1) apparatus for protection and prevention of deformities, (2) corrective apparatus, and (3) functional apparatus.

Apparatus for protection and prevention of deformities is that aiming to keep the affected segments in physiologic positions, that is, it maintains the affected joints in a neutral position. It is used especially when the changes in the elasticity and contractility in the muscles are beginning (contracture or spasm, paresis or paralysis) to cause a muscular unbalance (convalescent stage).

Corrective apparatus is that used in preventing incipient skeletal deformities.

Functional apparatus is that which facilitates or permits functioning of the locomotive system and presents the most complicated problem regarding its manufacture and adaptation, trying the knowledge of the physician and the ability of the manufacturer.

Orthopedic apparatus begins to be functional when it stabilizes a segment to facilitate the activity of the person; its characteristics are perfected when it allows the paretic segment to have passive or active motion, and it reaches the ideal in function when by means of mechanical attachments it cooperates in the execution of active motion.

The basic principles of this functional appartus are: (1) to maintain the postural alignment of the body, (2) to preserve the maximum of muscle-skeletal function, (3) to prevent deformities, (4) to reestablish the stability with the weight of the body, (5) to be as light as possible in order not to force weakened muscles, and, (6) to substitute for the nonrecovered function by means of the apparatus.

The experience of the hospital with orthopedic apparatus in the treatment of poliomyelitis is discussed. At the same time, a description is made of the modifications or innovations that the necessities, the means, and the type of patients have imposed.

FROM THE AUTHORS' SUMMARY.

Chronic Infectious Diseases

EFFECTS OF INTRATHECAL TUBERCULIN AND STREPTOMYCIN IN TUBERCULOUS MENINGITIS: AN INTERIM REPORT. H. V. SMITH and R. L. VOLLUM, Lancet **2**:275 (Aug. 19) 1950.

When tuberculous meningitis is treated for a long time with intramuscular and intrathecal injections of streptomycin, between 50 and 60% of patients recover. But the mortality has not been further reduced, and in the individual case the prognosis remains uncertain.

The authors studied the cerebrospinal fluid during the first few weeks of streptomycin therapy and noted that the fluctuations in the cell count and protein level suggest that tuberculin is liberated into the cerebrospinal fluid.

In patients without meningitis but sensitized to tuberculoprotein, as shown by a positive reaction in the Mantoux test, an intrathecal injection of minute amounts of the purified protein derivative of tuberculin U. S. P. produces fever and vomiting and also a pleocytosis and rise in protein in the cerebrospinal fluid indistinguishable from the changes seen in patients with untreated tuberculous meningitis. Rather larger doses reproduce the changes seen in cells and protein when tuberculous meningitis is treated with streptomycin.

In certain cases of tuberculous meningitis in which streptomycin has failed to effect a cure, the cerebrospinal fluid becomes sterile, and fluctuations in the cells and protein disappear. In two such cases the intrathecal injections of purified protein derivative of tuberculin given when the patients were apparently moribund reproduced the rise in cells and protein seen typically during the first few weeks of streptomycin therapy. In both patients these injections were followed by a totally unexpected recovery, which appears to be complete over a year after treatment was first begun.

Intrathecal injections of purified protein derivative of tuberculin U. S. P. were given in a third case in which the full decerebrate state had developed. The illness was prolonged for six months, and a definite objective improvement was seen, though this never amounted to a useful recovery. At necropsy no exudate was seen microscopically surrounding the brain stem.

In all three cases severe exudate must have been present and have resolved after the purified protein derivative of tuberculin was given. The authors suggest that the effects of tuberculin should be further explored now that any consequent spread of infection can be controlled by giving streptomycin at the same time.

Combined treatment with intrathecal injection of purified protein derivative of tuberculin and streptomycin has been begun by Smith and Vollum in seven other cases selected as having a bad prognosis. It is still too early to assess the results, but there has been only one death in the first three months of treatment. This compares favorably with the results on streptomycin alone.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

THE TYPES OF TUBERCULOUS PATHOLOGY FOUND IN CHILDREN UNDER THREE YEARS OF AGE AND WITH A KNOWN SOURCE OF CONTAGION. G. CROCCO and M. SAGGESE, Pediatria **61**:359 (May) 1953.

Crocco and Saggese report a study on 58 children under 3 years of age who had a known source of tuberculous infection. The source of infection was the father in 21 cases, the mother in 21 cases, both parents in 2 cases, a brother in 2 cases, a grandparent in 3 cases, an uncle in 3 cases, and the contact was from nonrelatives in 6 cases. The allergic type of tuberculosis was found in 36 children, early primary lesions in 15 children, disseminated basal lesions in 4 children, and early localized lesions in 3 children. The older the child the greater was the incidence of the allergic type.

HIGGINS, Boston.

TUBERCULIN ALLERGOMETRY AND THE SEDIMENTATION RATE: THE UNRELIABILITY OF ALLERGOMETRY IF TWO CONTEMPORANEOUS INTRADERMAL TESTS ARE MADE. G. VALLERINO and M. CAJATI, Pediatria **61**:425 (May) 1953.

The curves of the allergic reaction to tuberculin and the blood sedimentation rate do not run parallel in tuberculous patients. An allergometric determination by the method of Copello-Conti has been found to be unreliable if more than one intradermal test is made at the same time.

HIGGINS, Boston.

RELATIONS OF EYE-FUNDUS EXAMINATION TO X-RAY OF THE HEAD AND THORAX IN TUBERCULOUS MENINGITIS. A. PACI, Riv. clin. pediat. **51**:801 (June) 1953.

The author reports 21 cases of tuberculous meningitis in which the examination of the fundus of the eye and thorax and skull radiography (with and without pneumoencephalon) were executed, and studies their relationships. He talks about the value of such examinations for diagnosis and therapy and enlarges particularly upon pneumoencephalography, explaining the accidents and damage that can be produced in the subject by its exploitation during active illness.

FROM THE AUTHOR'S SUMMARY.

SKIN REACTIONS TO HISTOPLASMIN AND COCCIDIOIDIN IN A GROUP OF CHILDREN IN TUSCANY. ALBERTO FOIS, Riv. clin. pediat. **51**:840 (June) 1953.

In 145 children living in Tuscany, the skin reactions to coccidioidin, histoplasmin, and tuberculin have been tested. In 31, positive responses to tuberculin were observed; none reacted to coccidioidin or to histoplasmin. Sixty-four chest x-ray examinations were performed, including 34 tuberculin negative; of the latter, 12 showed evidence of pulmonary calcifications.

FROM THE AUTHOR'S SUMMARY.

THE ANTITUBERCULOUS ACTION OF OXYTETRACYCLINE (TERRAMYCIN). F. RAGAZZINI and P. MOGGI, Riv. clin. pediat. **51**:845 (June) 1953.

The authors refer the results obtained from their personal researches concerning the actions exercised by oxytetracycline (Terramycin) in vitro and in vivo on Mycobacterium tuberculosis and on blood liquid levels after oral administration. After having consulted the data obtained from medical literature, they conclude by not justifying exclusive oxytetracycline treatment in human tuberculosis.

FROM THE AUTHORS' SUMMARY.

LIVER CHANGES IN CONGENITAL SYPHILIS IN THE INFANT. JORGE ROSSELOT, HERMANN NIEMEYER, and JULIA ESPINOZA, Rev. chilena pediat. **24**:179 (June) 1953.

The authors stress the clinical, biological, and histological bases on which it is possible to diagnose liver involvement in congenital syphilis in infancy. In 64% of 70 cases they found a clinical hepatomegaly; in 47% of these it was associated with splenomegaly. In three cases there was jaundice that could be explained by the syphilitic infection in premature infants with general involvement. One of these cases was of a syphilitic newborn infant only 6 days old, in whom prolonged jaundice developed with clay-colored stools and in whom a liver biopsy established the diagnosis of cirrhosis.

In 64% of the cases liver flocculation tests were greatly altered. They give the data on the important histological findings in 40 congenital syphilitics who died, and they stress that often the lesions found are nonspecific. In 12 cases a liver biopsy was done, which they consider an aid to diagnosis.

FROM THE AUTHORS' SUMMARY.

ELECTRO-ENCEPHALOGRAPHY IN THE COURSE OF BACILLARY (TUBERCULOUS) MENINGITIS IN THE INFANT: REPORT OF THIRTY-ONE CASES. M. TURNER, R. VIGOUREUX, A. ROGERS, and M. TAURY, Rev. chilena pediat. **24**:192 (June) 1953.

The authors make correlations of electroencephalographic, clinical, and anatomical data in 31 cases of tuberculous meningitis in children. The findings on electroencephalogram consist of the following: bilateral and synchronic abnormalities, (*a*) amount of slow activity in relation to age, (*b*) bursts of hypersynchronous slow waves (theta and delta) and focal discharges, (*a*) "sharp-waves" and "spike-and-waves" in the temporal regions, (*b*) asymmetry between homologous areas.

These disorders seen on electroencephalogram are correlated with anatomical findings controlled by operation or necropsy. They correspond to preferential lesions of the optochiasmatic and basal regions of the brain and meningocortical lesions of the convexity.

FROM THE AUTHORS' SUMMARY.

SENSITIVITY OF KOCH'S BACILLUS IN VITRO TO ANTIBIOTICS. HERNAN GARCES, Rev. chilena pediat. **24**:202 (June) 1953.

The in vitro action of antituberculosis drugs of common clinical use on various strains of Koch's bacillus is studied. A wide margin of sensitivity to the drugs is encountered in the different strains in relation to the virulence. It was possible to demonstrate potentialization when associations of drugs were employed notably on the most virulent forms. After a certain time had elapsed, it was possible to demonstrate a diminishing effectiveness of the antibiotic, also in relation to the virulence of the strain. The antibiotic action appears to be prolonged with certain drug associations.

FROM THE AUTHOR'S SUMMARY.

SERUM PROTEINS IN CONGENITAL SYPHILIS. HELMUT KARTE, Monatsschr. Kinderh. **101**:47 (Feb.) 1953.

Sixty electrophoretic serum-protein fractionations were performed in 14 infants with congenital syphilis before and during treatment.

In eight infants with the visceral form of congenital syphilis, markedly decreased albumen, increased gamma globulin, and normal or slightly increased beta globulin values were found. In three patients the albumen value was low without significant increase in gamma globulin. Serum-protein changes were thought to be due to impaired liver function and became normal during antisyphilis treatment.

Two syphilitic infants with the nephrotic syndrome showed marked gamma globulin increases. Four young children with latent congenital syphilis and seven mothers with acquired latent syphilis were found to have normal or slightly abnormal electrophorecti serum-protein values.

No relationship between magnitude of serum-protein abnormalities and cardiolipin titers was demonstrated. Prognosis in infants with severe dysproteinemia was no more unfavorable than in those with only moderate changes.

HARTENSTEIN, Syracuse, N. Y.

TEST OF THE PERMEABILITY OF THE BLOOD: SPINAL FLUID BARRIER IN THE COURSE OF TUBERCULOUS MENINIGTIS. S. A. CHAROKOPOS, J. C. MARGARITIS and A. ZAFIRIS, Acta pædiat. **42**:184 (Sept.) 1953.

The authors have studied the permeability of the blood-liquid barrier in children with tuberculous meningitis for sodium bromide, fluorescein sodium (Uranine), streptomycin, and penicillin, and its relation with the chemical and cytological composition of the cerebrospinal fluid and the clinical symptomatology of the illness. They have come to the conclusion that this study can help with treatment and prognosis of tuberculous meningitis.

FROM THE AUTHORS' SUMMARY.

TUBERCULIN-SENSITIVITY AND ANTIBODIES (AGGLUTININS) AFTER BCG-VACCINATION. R. LAGERCRANTZ and H. ENELL, Acta paediat. **42**:316 (July) 1953.

Two hundred forty-eight healthy school children who had been BCG-vaccinated three months to five years previously were tuberculin tested, and blood samples were drawn for hemagglutination tests (Middlebrook-Dubos). All except 6.1% were tuberculin-positive and there was only an insignificant fall of tuberculin sensitivity in the groups vaccinated three to five years earlier. Sixty-five per cent of the children had a hemagglutination titer of at least 1:8. The titers of the children vaccinated three months previously were significantly higher than the titers of the children, who had been vaccinated earlier. There was no correlation between high tuberculin sensitivity and an elevated titer, nor between a newly increased sensitivity and a higher titer.

FROM THE AUTHORS' SUMMARY.

FURTHER STUDIES ON HEMAGGLUTINATION-TESTS IN PRIMARY, SECONDARY AND TERTIARY TUBERCULOSIS. R. LAGERCRANTZ and I. WOLFROM, Acta pædiat. **42**:330 (July) 1953.

Out of 63 patients with primary tuberculosis, 72% showed a titer value of at least 1:8, using Middlebrook-Dubos' technique, compared with 78% out of 83 patients with secondary, and 84% out of 144 patients with tertiary tuberculosis. There is no certain correlation between the clinical picture and the titer values except that all cases of severe meningitis with bad general state of health have shown no increase, and these cases had a bad prognosis. The practical use of the reaction is limited, but it can give some interesting information on immunity reactions in tuberculosis, which is commonly considered to be a tissue-bound reaction. A new approach with purified antigens might be valuable.

FROM THE AUTHORS' SUMMARY.

LABORATORY DIAGNOSIS AND EPIDEMIOLOGY OF TOXOPLASMOSIS. SVEN, GARD, Nord. Med. **45**:352 (March 7) 1951.

Toxoplasma can often be found in great numbers in the cerebrospinal fluid of infants with congenital toxoplasmosis. Ventricular puncture is to be recommended, as occlusion of the aqueduct not infrequently prevents the escape of the parasite into the lumbar fluid. Definite identifica-

tion of the parasite is obtained by inoculation into mice and subsequent serological analysis of the strain thus isolated. In all cases of toxoplasmosis serological tests should be performed. The neutralization test in the rabbit skin is unreliable and often difficult to interpret. The method of choice is the dye test of Sabin and Feldman in combination with the complement fixation test. In the acute phase of infection the dye test titer usually rises to 1:1,000 or more. The lowest peak value observed by Gard was 1:250. In most cases a decrease is observed after three to six months. The toxoplasmin skin test shows good agreement with serological reactions. The sensitivity of the test, however, is not correlated with the level of serum immunity. In about 10% of the serologically positive cases the skin test was negative. The toxoplasmin skin test is therefore of limited diagnostic value but might be useful in an epidemiological survey. An examination of 1,894 persons in Stockholm showed that 45.6% of the men and 46.4% of the women had a positive toxoplasmin test. In children of preschool age positive reaction were observed only in those with clinical signs of congenital toxoplasmosis. From the age of 5 years the incidence progressively increased, reaching about 40% at 20 years. A similar investigation in northern Sweden showed less than 10% positive reactions in adults, and one-half of the persons with positive reactions had moved into that region from other parts of the country. This different geographical distribution of latent toxoplasmosis indicates an extrahuman reservoir of infection. The author suggests insect transmission as a mode of spreading the disease.

WALLGREN, Stockholm, Sweden.

TUBERCULIN ALLERGY AFTER BCG VACCINATION. HERBERT ENELL, Nord. med. **46**:1072 (July 11) 1951.

The author reports a follow-up study of 625 BCG-vaccinated school children. Out of 104 children BCG vaccinated in 1945 only 5 had become tuberculin-negative in 1950. Clinical tuberculosis had developed in none of the children who were BCG vaccinated. Of the 475 nonvaccinated children 26 had become tuberculin-positive after starting school, and 13 of them had had clinical symptoms of tuberculosis. The examined group consisted of 1,100 pupils.

WALLGREN, Stockholm, Sweden.

BRONCHIAL TUBERCULOSIS IN CHILDREN. KJELL ROGSTAD, Nord. med. **47**:46 (Jan. 11) 1952.

Bronchoscopic evidence of gland perforation was found in 6 out of 41 children examined for primary pulmonary tuberculosis. Granulations and/or edema, with stenosis in the segmental bronchi with pus, were found in 25 other children. None of the patients had progressive caseous pneumonia, but in at least four of them phthisis developed in later childhood. Treatment should be conservative with *p*-aminosalicylic acid or streptomycin. Surgery, such as thoracic resection or lobectomy, may be necessary because of bronchiectasis or phthisic progression.

WALLGREN, Stockholm, Sweden.

Diseases of Blood, Heart and Blood Vessels, and Spleen

POSSIBLE PATHOGENIC MECHANISMS RESPONSIBLE FOR HUMAN PERIARTERITIS NODOSA: AS SUGGESTED BY THE OCCURRENCE OF TWO INSTANCES OF THIS DISEASE IN ASSOCIATION WITH GLOMERULONEPHRITIS. G. F. KIPKIE and D. S. JOHNSON, A. M. A. Arch. Path. **51**:387 (April) 1951.

There are reported the cases of a 7-year-old and an 11-year-old, both white boys, who died following symptoms of renal failure. History in both cases revealed the use of sulfonamides in the past. At autopsy, both patients were found to have had glomerulonephritis, acute in one and subacute in the other. In addition, lesions of periarteritis nodosa were found generalized in one case and confined to the pulmonary arteries in the other. The latter case was also one with the cardiac malformation of Eisenmenger's complex. The possible mechanisms involved in the production of the periarteritis put forth by the authors include the following: (1) The periarteritis was due to a pathogenic process which was the same as that responsible for the glomerulonephritis; (2) it was the result of an associated hypertension, or (3) it was an allergic reaction

to a sulfonamide compound. The authors conclude that the data do not permit assigning the periarteritis nodosa occurring in these cases specifically to the operation of any one of the suggested mechanisms. They believe that probably hypersensitivity was most important but that it probably acted along with some unknown factor.

HAW CHAN, San Francisco.

CONGENITAL INTERATRIAL COMMUNICATIONS: CLINICAL AND SURGICAL CONSIDERATIONS WITH A DESCRIPTION OF A NEW SURGICAL TECHNIC: ATRIO-SEPTO-PEXY. C. P. BAILEY; D. F. DOWNING; G. D. GECKELER, W. LIKOFF; H. GOLDBERG; J. C. SCOTT; O. JANTON, and H. P. REDONDO-RAMIREZ, Ann. Int. Med. **37**:888 (Nov.) 1952.

The authors trace in a lucid manner the embryology, pathology, and pathologic physiology of interatrial lesions. The clinical picture, including incidence, symptoms, physical findings, course, and diagnostic measures, is discussed in detail.

Corrective surgical procedures, such as those described by Cohn, Murray, Martin and Essex, Swan, Hufnagel, and Doddrill, are critically evaluated. The faults of these procedures are found to be thrombus formation, interference with atrial flow, introduction of foreign bodies, production of arrhythmias, and actual failure to close the defect. Of these procedures Swan's technique seems to be of greatest value, particularly for patients in very poor operative condition.

A new procedure, which involves suturing the right atrial wall to the margin of the defect, is illustrated. The evolvement of this procedure in nine cases of atrial surgery is described. This technique was attempted in six cases, four of which had satisfactory results. Two operative deaths occurred in patients with very advanced disease. This is the first report of true intracardiac suturing for relief of cardiac defects.

ETTINGER, Chicago.

THE NEWER HEMATINICS, THEIR USE AND ABUSE. WILLIAM F. LUTTGENS, California Med. **77**:369 (Dec.) 1952.

The newer hematinics are merely refinements of preexisting forms of treatment, but they have aided particularly in a better understanding of the deficiency states. The intrinsic factor of Castle has not been isolated from the gastric juice, and the interrelationships of this substance with the extrinsic factor (vitamin B_{12}) and folic acid have not been defined at this time. Vitamin B_{12} appears to be the active principle of refined liver extract and alone is probably adequate treatment for pernicious anemia. The other varieties of megaloblastic anemia may result from deficiency of vitamin B_{12} or folic acid, although generally treatment with the latter brings about complete and lasting remission.

The use of multihematinics and multivitamin preparations containing folic acid is to be condemned, particularly because of the possibility of their obscuring anemia and thwarting the diagnosis of pernicious anemia until neurologic complications have taken place.

Saccharated iron oxide is a relatively safe preparation for intravenous administration, but the indications for its use are few. Because the body has no mechanism for iron excretion, only the amount of iron necessary to make up a deficiency should be given, although there is no definite evidence that hemochromatosis results from overdosage.

PRYOR, Redwood City, Calif.

CONGENITAL HEART DISEASE IN CYANOTIC CHILDREN. WILLIS J. POTTS, California Med. **78**:101 (Feb.) 1953.

Cyanosis is often the only apparent symptom of congenital heart disease for which a child is brought to a physician. Some of the more frequent anomalies can be diagnosed from this and other symptoms by a general practitioner. Squatting after exertion is a sign of the tetralogy of Fallot; severe disability with relatively mild cyanosis may indicate pure pulmonary stenosis. A brisk, short, rasping systolic murmur is characteristic of these conditions and of tricuspid atresia.

The tetralogy of Fallot is further characterized by a boot-shaped heart, not greatly enlarged, and right axis deviation on electrocardiograms. Typically, the lung fields are clear. The author's

treatment of choice is aortic-pulmonary or subclavian-pulmonary anastomosis as indicated, preferably done after the child is 3 years old if the condition is not so severe as to necessitate earlier operation.

Pure pulmonary stenosis, which in some cases cannot be distinguished from the tetralogy of Fallot except by cardiac catheterization and angiocardiography, may in more typical cases be diagnosed by convexity rather than concavity in the pulmonary segment and by differences in electrocardiograms. An expanding valvulotome is used to open the stenosed pulmonary valve, which is then dilated.

A systolic murmur, a round heart, and a left axis deviation are usually found in tricuspid atresia. Shunt operations performed for relief of this condition may lead to later heart failure because of the devious rerouting of blood through the heart.

The operations here outlined and others are statistically evaluated.

PRYOR, Redwood City, Calif.

MULTIPLE LESIONS OF BONE IN LETTERER-SIWE DISEASE: REPORT OF A CASE WITH CULTURE OF PARACOLON ARIZONA BACILLI FROM BONE LESIONS AND BLOOD, FOLLOWED BY RESPONSE TO THERAPY. RICHARD H. FISHER, J. Bone & Joint Surg. **35-A**:445 (April) 1953.

Fisher presents a detailed report on a case of Letterer-Siwe disease which was proved to be due to paracolon Arizona bacilli and which was cured with antibiotics. It appears to be the first reported case in which a definite organism has been associated with Letterer-Siwe disease or a process closely resembling it. Four other cases of Letterer-Siwe disease cured by antibiotics have been reported in the last two years.

The role of the paracolon bacillus could be interpreted in three ways. First, as a fortuitous complication of Letterer-Siwe disease; second, as the cause of a paracolon osteomyelitis with multiple lesions simulating Letterer-Siwe disease, and third, as the cause of a case of Letterer-Siwe disease. The fact that complete healing followed use of antibiotics indicates that the disease was caused by the organism; to assume that this was osteomyelitis would entail the assumption that there are two similar diseases, one of which is due to infection and the other not. It has, however, been long suspected that Letterer-Siwe disease is due to infection. This case is the first in which such a relationship has been proved.

Treatment was with chloramphenicol and oxytetracycline (Terramycin.) Penicillin was of no value.

MCGUIGAN, Evanston, Ill.

THROMBOSIS OF THE INFERIOR VENA CAVA AND RENAL VEINS WITH HEMORRHAGIC RENAL INFARCTION IN INFANCY. DAN FERIOZI, E. CLARENCE RICE, WILLIAM F. BURDICK, and FRANCIS J. TROENDLE, J. Pediat. **38**:235-241 (Feb.) 1951.

Thrombosis of the inferior vena cava and renal veins developed in a 4-month-old male infant, with renal infarction secondary to an acute ileocolitis. This case surprisingly had hypertension, a rare finding.

MACDONALD, Pittsburgh.

SPATIAL VECTORCARDIOGRAPHY IN CHILDREN: AN ANALYSIS OF HIGH R WAVES IN RIGHT-SIDED CHEST LEADS. RICHARD P. LASSER and ARTHUR GRISHMAN, J. Pediat. **39**:51-60 (July) 1951.

Normal children under the age of 8 years were compared with children of the same age with right ventricular hypertrophy associated with congenital heart disease. Both groups had similar electrocardiograms, with prominent R waves in Lead V_1. However, spatial vectorcardiograms of these two groups differed. In the normal group there was displayed a horizontal plane vectorcardiogram, with (1) a large initial anterior loop mostly counterclockwise in rotation, (2) inscription of the centripetal limb posterior to the centrifugal limb, and (3) inscription of most of the loop anteriorly.

Vectorcardiograms of children with congenital heart disease showed a small initial loop clockwise in rotation and inscription of the centripetal limb anterior to the centrifugal limb.

MACDONALD, Pittsburgh.

AMELIORATIVE EFFECT OF ANTIBIOTICS ON NONLIPOID RETICULOENDOTHELIOSIS (LETTERER-SIWE DISEASE) IN IDENTICAL TWINS. HOWARD R. BIERMAN, JONATHAN T. LANMAN, and KENNETH DOD, J. Pediat. **40**:269-284 (March) 1952.

Bierman and his colleagues report the simultaneous development of Letterer-Siwe's disease in identical male twins. The administration of a combination of chlortetracycline (Aureomycin), chloramphenicol (Chloromycetin), streptomycin, and penicillin was followed in each twin by clinical improvement, with disappearance of hepatosplenomegaly and lymphadenopathy, with regression of anemia and roentgen evidence of healing, and with disappearance of osteolytic lesions. Although both children now appear well, signs of residual disease and sequelae remain.

MACDONALD, Pittsburgh.

TREATMENT OF ACUTE LEUKEMIA IN CHILDREN WITH AND WITHOUT FOLIC ACID ANTAGONISTS. H. G. PONCHER, H. A. WAISMAN, J. B. RICHMOND, O. A. HORAK, and L. R. LIMARZI, J. Pediat. **41**:377 (Oct.) 1952.

The authors discuss Aminopterin as a folic acid and citrovorum factor antagonist in respect to the enzymes concerned in protein metabolism associated with cellular maturation In this report, folic acid antagonists, Aminopterin, and A-Methopterin have provided clinical improvement and prolonged life in the majority of cases.

MACDONALD, Pittsburgh.

FURTHER STUDIES ON THE BLOOD OF CHILDREN WITH CYANOTIC HEART DISEASE WITH SPECIAL REFERENCE TO THE HEMOGLOBIN. FORREST H. ADAMS and SHIRLEY C. CUNNINGHAM, J. Pediat. **41**:424 (Oct.) 1952.

Anoxia has a somewhat different effect in the fetus, in the person living at high altitude, and in the patient with cyanotic heart disease. Children with cyanotic heart disease have polycythemia. This is not associated with changes in the size of the red blood cells, fragility, or with the carbonic anhydrase content. There is no significant increase in reticulocytes, but there is a significant decrease in the number of platelets. There are no changes in the bone marrow. Alkali denaturation tests on the hemoglobin from such cyanotic children show that the hemoglobin is adult rather than fetal in character.

MACDONALD, Pittsburgh.

A SUMMER CAMP FOR CHILDREN WITH HEART DISEASE. LUCILLE J. ROSS, J. Pediat. **43**:67 (July) 1953.

Ross describes a summer camp for children with heart disease. The camp had a special psychological value for the children and their parents. As Dr. Ross points out, a certain number of her campers should have been acceptable at any normal summer camp.

This is a detailed report of a pioneer adventure.

MACDONALD, Pittsburgh.

ANTIBIOTIC THERAPY OF BACTERIAL ENDOCARDITIS: II. CURRENT TRENDS IN THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS. JOSEPH E. GERACI, Minnesota Med. **35**:861 (Sept.) 1952.

Clinical data on 46 cases of endocarditis are presented. In 34 cases, streptococci were isolated. Hemophilus was isolated in one case. Sensitivity tests should be done on all organisms. In 9 cases penicillin alone was used, and in 37 cases combined therapy was used.

The dosage and duration of treatment for this disease must be determined by the causative organism and its apparent sensitivity to the antibiotic used.

Fifteen consecutive cases of endocarditis caused by penicillin-sensitive streptococci, in which short-term combined therapy with penicillin and dihydrostreptomycin was used, are summarized. It would appear that the use of 2,000,000 units of penicillin and 2 gm. of dihydrostreptomycin per day administered intramuscularly in divided doses every 6 to 12 hours for 10 to 14 days is adequate treatment in these cases.

STOESSER, Minneapolis.

ASYMPTOMATIC ISOLATED VALVULAR PULMONARY STENOSIS: DIAGNOSIS BY CLINICAL METHODS. S. GILBERT BLOUNT JR., SEIICHI KOMESU, and MALCOLM C. MCCORD, New England J. Med. **248**:5 (Jan. 1) 1953.

Isolated pulmonary stenosis is not a rare congenital cardiac anomaly but one of the commoner types. Six cases of isolated pulmonary valvular stenosis are reported and discussed, with principal emphasis on clinical evaluation.

This entity exists in an apparently mild form, the patient appearing normal in all respects, and the correct diagnosis may be established on clinical grounds with a high degree of accuracy.

Physical examination reveals a thrill and a harsh systolic murmur that is of maximum intensity in the first or second left intercostal space and that is almost always louder in the first than in the third left intercostal space. The second heart sound in the first and second left intercostal spaces is usually decreased in intensity to absent, and, though it may be of normal intensity, it is always pure. The electrocardiogram may be normal or may show evidence of hypertrophy of the right ventricle of varying degree. Fluoroscopic examination is of great importance in indicating the correct diagnosis. The poststenotic dilatation is always present, and a disproportion exists between the amplitude of the pulsation over the main pulmonary arteries. The over-all vascularity of the lung fields may be within normal limits, although it is frequently slightly decreased toward the periphery. The heart may be within normal limits in size, or there may be evidence of hypertrophy of the right ventricle. The right auricle may be enlarged. Enlargement of the right auricle with a normal-sized heart in a patient without symptoms is considered to be a finding of significance and to suggest the diagnosis of isolated pulmonary stenosis. Cardiac catheterization confirms the clinical diagnosis but is of greatest importance in determining the management of the patient. Finally, it is again emphasized that the asymptomatic patient has a process that, unless recognized early and corrected surgically, may be fatal.

WHEELOCK, Denver.

THE USE OF DIGITALIS IN INFANTS AND CHILDREN: A CLINICAL STUDY OF PATIENTS IN CONGESTIVE HEART FAILURE. ALEXANDER S. NADAS, ABRAHAM M. RUDOLPH, and JOHN L. REINHOLD, New England J. Med. **248**:98 (Jan. 15) 1953.

The response to digitalis of 41 infants and children in congestive heart failure is described. The incidence of nonrheumatic causes of congestive failure in the young is discussed. Congenital heart disease and "primary myocardial disease" are the two conditions encountered frequently in this group.

Congenital heart disease and paroxysmal tachycardia are most likely to cause failure within the first six months of life. Patients with myocardial disease and rheumatic fever are most likely to fail after 2 years of age.

The most satisfactory response to digitalis was obtained in patients with myocardial disease, paroxysmal tachycardia, and rheumatic carditis. The least satisfactory results were obtained in children with congenital heart disease, especially those with cyanosis.

No accurate estimates of the level of digitalization could be obtained by the electrocardiogram. The most consistent electrocardiographic change following digitalis administration was shortening of the Q T_c.

A satisfactory dosage schedule may be obtained if the age and weight of the patients are taken into consideration. Patients under 2 years of age need 0.02 to 0.03 mg. of digitoxin per pound of body weight for digitalization. Children over 2 years respond favorably to dosages ranging between 0.01 and 0.02 mg. per pound of body weight. Electrocardiographic evidence of digitalis intoxication was found most commonly in patients who responded most favorably to the glycoside.

WHEELOCK, Denver.

ALYMPHOCYTOSIS. W. L. DONOHUE, Pediatrics **11**:129 (Feb.) 1953.

This study describes an unusual fatal disease in a child, which was characterized by fever, hepatomegaly, splenomegaly, and a leucopenia in which the striking feature was lymphocytopenia.

Postmortem examination revealed a widespread "idiopathic" atrophy and necrosis of the lymphoid tissue. An associated finding was an extensive Candida infection of the esophagus.

FROM THE AUTHOR'S SUMMARY.

HODGKIN'S DISEASE: A STATISTICAL REPORT. R. W. DICKENHORST and W. H. CARROLL, Radiology **59**:221-228 (Aug.) 1952.

Seventy-two cases with proved incisional histopathological diagnosis are included in the report. The youngest patient was 4 years old. Six patients were found in the first decade and 10 in the second decade. The ratio of males to females was 2:1. Of 42 patients representing all stages of the disease prior to 1947, 42.8% lived less than six months. Only 14.3% survived five years or longer. The over-all picture, according to the authors, indicates that there has been an upward trend in survivals throughout the past 19 years and that the relatively intensive irradiation of the past 10 years is far superior to the moderate therapy given earlier.

ANSPACH, Riverside, Ill.

ROENTGENOLOGIC CONSIDERATIONS IN THE DIAGNOSIS OF CONGENITAL TRICUSPID ATRESIA. SUMNER N. MARDER, WILLIAM B. SEAMAN, and WENDELL G. SCOTT, Radiology **61**:174 (Aug.) 1953.

Regular roentgenograms frequently reveal unequivocal evidence of pulmonary stenosis by the reduction in caliber of the pulmonary arteries, but angiography, though limited in value, is essential for the diagnosis of the fundamental malformations. Extensive bronchial collateral circulation (numerous small vessels) may obscure somewhat the clear lung fields, especially in older children. The presence of a right aortic arch favors a diagnosis of tetralogy of Fallot, but it does not exclude tricuspid atresia. The contour of the cardiac shadow rarely permits a differentiation of these two conditions.

In tricuspid atresia contrast visualization shows opacification in this sequence: right auricle, left auricle, and left ventricle. A nonopaque triangle seen on regular anteroposterior roentgenograms formed at the diaphragm of the right auricle and left ventricle is a helpful diagnostic sign.

The authors believe that the electrocardiographic findings of left axial deviation or left ventricular hypertrophy as clinically used in differentiating tricuspid atresia from other anomalies presenting chronic cyanosis and pulmonic stenosis have limited value in many cases. These findings arouse a desire for more anatomical data and constitute the indications for angiocardiography. A review of the roentgenological findings in eight cases is given.

ANSPACH, Riverside, Ill.

RELATION OF BLOOD PRESSURE TO WEIGHT, HEIGHT, AND BODY SURFACE AREA IN SCHOOLBOYS AGED ELEVEN TO FIFTEEN YEARS. LEO HAHN, Arch. Dis. Childhood **27**:51-52 (Feb.) 1952.

A study of blood pressure in 1,417 schoolboys, aged 11 to 15 years, is presented.

Mean, standard deviation, range, and percentage distribution of systolic and diastolic blood pressure were determined, the correlation between systolic and diastolic blood pressure ascertained, and formulae established from the regression equations.

The study reveals (1) that there is a wide variation of systolic and diastolic blood pressure which becomes more marked once puberty starts; (2) that systolic and diastolic blood pressure "spurts" with the beginning of puberty, i. e., at the same time as the third period of acceleration of growth, and (3) that 78.1% of the 11 5-year-old boys and 70.5% of the 12 5-year-old boys have a systolic blood pressure between 90 and 119 mm., whereas three-quarters of the 13½- to 15½-year-old boys exhibit a systolic blood pressure between 100 and 129 mm. Of the 15 5-year-old boys, 62.3% have a systolic blood pressure between 110 and 139 mm. About three-quarters of the 11-, 12-, and 13-year-old boys have a diastolic blood pressure between 50 and 69 mm. Hg, whereas 72.5 and 81.8% of the 14- and 15-year-old boys show a diastolic blood pressure within the range of 60 to 79 mm. Hg. There is a statistically significant correlation between systolic and diastolic blood pressure.

Generally each boy has the blood pressure of his physiological age.

The correlation of blood pressure, weight, and body surface area permits the prediction of the normal blood pressure for the individual boy from the respective regression equation, and formulae to this effect are presented.

KELLY, Milwaukee.

APLASTIC CRISIS IN CONGENITAL HÆMOLYTIC ICTERUS (ACHOLURIC JAUNCICE): A DESCRIPTION OF THREE CASES. J. MARGOLIS, Med. J. Australia **1**:100 (July 18) 1953.

Three cases are discussed, the patients in all of them suffering from congenital hemolytic icterus and demonstrating aplastic crises. All were members of the same family. Bone marrow studies in one case showed definite aplasia, and it is felt that the term aplastic crisis should be used, rather than hemolytic crisis.

McGUIGAN, Evanston, Ill.

THE SICKLE CELL TRAIT IN CAPE COLOURED PERSONS. S. R. ESRACHOWITZ, S. FRIEDLANDER, G. RADLOFF, and S. SAUNDERS, South African M. J. **26**:239 (March 22) 1952.

Studies of 1,555 colored natives revealed a sickling trait to be present in 0.58%.

McGUIGAN, Evanston, Ill.

ESSENTIAL LYMPHOCYTOPHTHISIS. EDUARD GLANZMANN and PAUL RINIKER, Ann. pædiat. **175**:1 (July-Aug.) 1950.

Based on the observation of two identical cases in early infancy, a fatal infection with Candida albicans is described under the name "essential lymphocytophthisis." This disease not only attacks the whole oropharynx but also stretches into the larynx and lungs, the whole length of the esophagus, stomach, and bowel. At an early stage there is a lymphopenia in the blood which toward the end goes down to a few per cent (alymphocytosis as an analogue to agranulocytosis). The lymphocytophthisis may be joined at the end by a panmyelopathy. Cases of lymphocytophthisis, on the one hand, and panmyelophthisis, on the other hand, in the same family point to a constitutional weakness of the blood-forming organs. In later stages large macular, morbilliform, septic exanthemata occur in batches.

The anatomical signs of essential lymphocytophthisis are loss of lymphocytes in the gut, in the lymph nodes, and in the spleen, and various degrees of proliferation ex vacuo in the reticular stroma which later on may show hyaline or fibrous degeneration. The disappearance of the Peyer plaques may leave sharply defined bare patches on the mucous membrane which macroscopically look like small cleaned ulcers. The shape and size of the lymph nodes are preserved, in spite of the complete absence of the lymphocytes, through the widening and proliferation of the cortical and medullary sinus. The lymphocytes are absent also in the spleen. The bone marrow becomes excessively aplastic toward the end. The megacaryocytes persist for a long time. Besides the constitutional factor leading to lymphocytophthisis, disturbance of intestinal resorption on account of an infection with Candida albicans producing necrosis has to be made responsible for the lethal course of the illness.

FROM THE AUTHORS' SUMMARY.

ON SERUM COPPER LEVEL AND ITS IMPORTANCE IN CHILDHOOD. E. STRANSKY, D. T. DAUIS-LAWAS, and I. L. LAWAS, Ann. pædiat. **179**:1 (July) 1952.

Serum copper level determinations were made in 144 children. It was found (1) that the copper level in Filipino children is extremely high, the average being 254.4 γ per 100 cc., which is contrary to the reported averages in the literature of slightly more than 100 γ per 100 cc.; (2) that the copper level of the serum is independent of the age and sex of the children, and (3) that the copper bears no relation to the hemoglobin level of the blood or to the serum iron level.

So long as we do not know more about copper metabolism and the role of copper in metabolism, no final comment on the importance of hypercupremia can be given. It is, however, probable that hypercupremia is a symptom of malnutrition, like hypoproteinemia, hyponatremia, low ascorbic acid level, and low alkaline serum phosphatase level—all found in Filipino children to a high degree. Hypercupremia may be due also to steatosis of the liver and to impairment of endocrine functions by atrophy of the respective glands.

FROM THE AUTHORS' SUMMARY.

CORRELATIONS OF MALFORMATIONS IN THE HUMAN HEART. FRITZ GLOOR, Ann. pædiat. **181**:99 (Aug.) 1953.

Gloor analyzes all the cases of cardiac malformations observed during the years 1938-1950 in the pathological institute of Basel University in an attempt to ascertain the frequency of the various forms of congenital heart malformations in the Basel area. One hundred twenty-two different malformations of the heart vessels, found during 16,565 autopsies, are analyzed and classified. With regard to the most frequently found malformations, the question of possible conditioned malformations in the heart is examined. The combination with malformations in other organs of the body is investigated, and the operability of some of the malformations is discussed.

FROM THE AUTHOR'S SUMMARY.

CONSTITUTIONAL PANMYELOPHTHISIS WITH MULTIPLE CHANGES (FANCONI'S SYNDROME). LUDWIG BARTA and ANTON TOSZEGI, Ann. pædiat. **181**:117 (Aug.) 1953.

The authors describe the 22d case of Fanconi's syndrome in world literature. In their patient were found all the classic signs of the syndrome, such as multiple developmental disturbances, pathological melanin pigmentation, hemorrhagic diathesis, aplastic anemia, and growth disturbances. The thrombasthenia they found together with observations made concerning coagulation and heparin content of the blood provide new facts toward a better knowledge of the clinical picture. Serial examinations of the bone marrow showed its destruction in detail.

FROM THE AUTHORS' SUMMARY.

DURATION OF THE LIFE OF THE RED BLOOD CORPUSCLE IN MEDITERRANEAN ANEMIA. G. FRONTALI and G. A. STEGAGNO, Helvet. pædiat. acta **6**:271 (Aug.) 1951.

The authors have studied the life span of red blood cells in Cooley's disease with the use of the technique of Ashby modified by Mollison.

The conclusions already communicated by the authors at the Academy of Medicine in Rome in May, 1950, and at the Sixth International Congress of Pediatry in Zurich in July, 1950, have been confirmed.

1. The life span of normal red cells transfused in normal subjects has been confirmed in term of 120 days.

2. The same life span has been found to be for normal red cells transfused in patients with Cooley's disease.

3. The life span of red cells of patients with Cooley's disease transfused in normal subjects has been shown reduced to 12 to 19 days, with steeply falling curves.

4. Still insufficient are the data concerning the life span of red cells in the parents of patients with Cooley's disease, showing the constitutional anomaly of the red cells.

FROM THE AUTHORS' SUMMARY.

REVERSIBLE MEGALOBLASTIC ANEMIA IN INFANCY. M. AMATO, Helvet. pædiat. acta **7**:291 (June) 1952.

Out of the numerous cases of reversible megaloblastic anemia in infancy seen at the Children's Clinic in Naples, the author selects and describes five cases which showed specific neurologic disturbances in addition to the usual clinical and hematologic manifestations. From his own observations and several analogous cases reported in the literature, the author arrives at the concept that in megaloblastic anemias of early infancy there can develop, before or during specific therapy with liver extract or folic acid, a hypertonic-dyskinetic neurological syndrome of variable intensity and extent, assuming a predominating extrapyramidal character and being functional, or reversible, in type.

As to the pathogenesis, the author advances the hypothesis that the neurological symptom, like the other manifestations of the underlying disease, should be considered as one among several phenomena produced by a catalytic disturbance of cell respiration.

FROM THE AUTHOR'S SUMMARY.

BLOOD PLASMA IN HEMOPHILIA. P. NOLF, Schweiz. med. Wchnschr. **80**:125-129 (Feb. 11) 1950.

The author previously attributed the diminished tendency of hemophilic plasma to coagulate to a lack of thrombozyme. Interesting experimental data are now given showing that a solution of thrombozyme freed from thrombogen and thromboplastin cannot accelerate the coagulation of plasma from a patient with hemophilia. A solution of plasmatic thromboplastin does accelerate the coagulation, and the opinion of others that a deficiency of thromboplastin characterizes hemophilic plasma is confirmed. This deficiency may perhaps be more qualitative than quantitative.

AMBERG, Rochester, Minn.

THE SYNDROME OF TAUSSIG AND BING. O. BAYER, Schweiz. med. Wchnschr. **81**:558 (June 9) 1951.

A Taussig and Bing syndrome was diagnosed in a 24-year-old man. The syndrome is discussed; it is characterized by the derivation of the aorta from the right ventricle and straddling of the dilated pulmonic artery above a high defect of the ventricular system and a hypertrophy of the right ventricle. Clinically it is similar to the Eisenmenger complex, from which it can be differentiated only on catheterization of the heart, with a greater degree of oxygen saturation in the pulmonic artery than in the arterial blood. The pressure in the pulmonic artery and the right ventricle is considerably elevated. It is to be noted specially that considerable cyanosis existed from time of birth.

AMBERG, Rochester, Minn.

EOSINOPHILIC LEUKEMOID AND RELATED DISEASES. TH. BAUMANN, Schweiz med. Wchnschr. **82**:940 (Sept. 20) 1952.

An eosinophilic leukemoid was observed in three instances—in an adult, a 22-day-old girl, and a boy about 7 years of age. In the case of the girl, a leucocytosis as high as 32,000 was registered, with an eosinophilia of 20%. Leucocytosis and eosinophilia persisted about five weeks. Hepatomegaly also regressed. A megalerythema and a scarlatiniform exanthema occurred and also a bronchiolitis and a bronchitis asthmatica. Furthermore, a vasomotor rhinitis was noted. In spite of a negative cutaneous test with human milk and the absence of a leucopenic index, an idiosyncrasy to mother's milk is assumed. The boy gave a history of Ascaris infestation over several years. The onset of the eosinophilic leukemoid was sudden. A leucocytosis of 31,000, with 24.4% eosinophiles, dropped on elimination of Ascaris in six days to 8,400, with 7% eosinophiles. In addition, urticaria, Quincke's edema, arthritic symptoms, transitory eosinophilic pulmonic infiltration, and a swelling of the liver were noted. All symptoms disappeared in nine days, being replaced for several days by a vasomotor rhinitis and an asthmatic bronchitis. Acute, subacute, and chronic eosinophilic leukemoid is regarded as an expression of an allergic-hypergic reaction.

AMBERG, Rochester, Minn.

TREATMENT OF CHILDREN WITH HEART DISEASE WITH DIGITOXIN. H. G. HANSEN, Schweiz. med. Wchnschr. **83**:36 (Jan. 10) 1953.

As with other digitalis preparations, the classic indication for the administration of digitoxin is insufficiency of the heart, with symptoms of stasis. In very acute stages of cardiac insufficiency, treatment is initiated by strophanthin for quicker action. In cases of chronic insufficiency prolonged treatment with small doses of digitoxin is recommended. The drug is given by mouth. It has proved satisfactory when the full therapeutic dosage was given in two to four days. It is recommended that an initial dose of 0.1 to 0.3 mg. be given to children of 3 to 6 years of age, 0.2 to 0.4 mg. to children of 6 to 10 years, and 0.3 to 0.5 mg. to children of 10 to 14 years. The dose should be divided in the first 24 hours. Regression of the frequency of the pulse is the first sign of the action of digitoxin, which is resorbed completely on oral administration. On the whole, the drug is tolerated very well, but careful observation of the patient under treatment is necessary, since the dose for optimal therapeutic effect is near the dose which elicits undesirable toxic side-effects.

AMBERG, Rochester, Minn.

SIDEREMIA AS AFFECTED BY FOOD. M. LATO, Clin. pediat. **33**:236 (April) 1951.

Ten normal breast-fed infants, 1 to 5 months old, had red blood cell counts averaging 5,000,000 and an average iron content of the blood serum of 140 γ per 100 cc. The iron content of the mother's milk almost paralleled that of the baby's blood serum, being 130 γ per 100 cc. Eight normal artificially fed infants, also 1 to 5 months of age, had red blood cell counts of about 4,000,000 and an average iron content of the blood of 90 γ per 100 cc. The iron content of cow's milk averages 75 γ per 100 cc. or only a little more than one-half that of mother's milk.

These findings demonstrate an advantage of breast feeding over artificial feeding in that a more normal iron metabolism is maintained.

HIGGINS, Boston.

ELECTROCARDIOGRAPHY AND HYPOPROTEINEMIA. L. SALMI, Clin. pediat. **35**:239 (April) 1953.

The electrocardiograms of 4 out of 22 patients with hypoproteinemia showed significant deviations from the normal, with extrasystoles, low voltage, and inverted waves. It seemed likely that the hypoproteinemia was the responsible factor, as the findings on electrocardiograms became normal after the hypoproteinemia had been corrected.

HIGGINS, Boston.

BLOOD PROCAINE-ESTERASE IN THE CHILD. T. RABAGLIA and G. CHIARI, Lattante **22**:269 (May) 1951.

Hazard had reported that normal blood has an enzyme which splits the procaine molecule; in patients with liver disease, this enzyme was diminished in amount of activity.

In the present study, blood procaine-esterase determinations were made on a group of 58 children. Children were found to react much more easily to the hepatic factor in the hydrolysis of procaine than did adults.

HIGGINS, Boston.

FIRST STUDIES ON TRANSVERSE AXIAL COMBINED WITH FRONTAL STRATIGRAPHY, WITH OR WITHOUT PNEUMOMEDIASTINUM IN THE TETRALOGY OF FALLOT BEFORE AND AFTER THE BLALOCK-TAUSSIG OPERATION AND IN EISENMENGER'S SYNDROME. R. BULGARELLI and E. OLIVA, Minerva pediat. **3**:275 (May) 1951.

It is possible by transverse axial and frontal stratigraphy to determine the location of the aortic arch, the degree of dextroposition of the aorta, and the size of the vena cava. Four cases of the tetralogy of Fallot were studied before and after the Blalock-Taussig operation. The x-ray pictures are shown to illustrate the markedly increased pulmonary circulation after the operation.

HIGGINS, Boston.

SICKLE-CELL ANEMIA IN GREECE. C. CHOREMIS, B. COSTANTINIDES, L. ZANNOS, and S. SKLAVOYNOY, Minerva pediat. **4**:149 (March 15) 1952.

Sickle-cell anemia was not reported in Greece until 1940. Since that time a number of other cases have been reported. During the last three years several cases were seen in the Children's Hospital of the University of Athens. All the patients came from the vicinity of Petromagoula, which with its surrounding villages has a population of about 6,000 inhabitants. This town and the neighboring villages were until recently one of the most malaria-stricken areas of Greece because of their location near a swamp. The swamp has now dried up, and this factor, together with the use of DDT, has rendered the neighborhood relatively free of malaria. Two surveys of the area have revealed many cases of sickle-cell anemia. Approximately 0.25% of the population had symptoms of sickle-cell anemia, and 15.6% of 1,016 inhabitants had sickling of the red cells, as found on examination of wet blood smears. This latter figure is almost double that found and reported for American Negroes (8.0%). The children with sickle-cell anemia showed a characteristic physical appearance not observed in patients with Cooley's anemia or in the reported cases of Negroes with sickle-cell anemia; the children had a flat nose, slightly prominent cheek bones, a wrinkling of the skin of the upper lip which displayed the gums above the teeth during a smile, and some degree of infantilism. The disease of sickle-cell anemia is also believed to be prevalent in another town, Gastouni in the Peloponnesus, similarly located near a swamp and with endemic malaria.

The disease is definitely hereditary. Cases of sickle-cell anemia occur only in children, both of whose parents are carriers of the sickle-cell trait. There is some possibility that there has been admixture of Negroes with Greeks in the past. Slaves and mercenary soldiers were frequently brought to Greece in ancient times; even as late as 1825 there was an Egyptian invasion of Greece. But it seems much more likely that some mutation of the genes had occurred in this area due to the factors of poverty, malaria, and intermarriage. The similarity of sickle-cell anemia and Cooley's anemia suggests that there have been two different but similar mutations, or one mutation which later split into two, to establish the bases of these two diseases. This article is written in English.

HIGGINS, Boston.

PERIARTERITIS NODOSA IN A CHILD. M. SCARZELLA and A. FORTINA, Minerva pediat. **4**:287 (April 15) 1952.

At the age of 4 years, a girl had a serious illness marked by impetigo, erysipelas, stomatitis, and gangrene of the terminal digital phalanges. Six years later she again became critically ill, this time with the characteristic clinical symptoms of periarteritis nodosa—septic fever, anemia, polymyositis, abdominal colic, renal difficulty, and typical nodules along the vessels of the arms and legs. The diagnosis was confirmed by biopsy of nodules. There were two short remissions before the child died.

HIGGINS, Boston.

ACUTE LEUKEMIA OF CHILDHOOD, INITIATED BY PSEUDOAPPENDICITIS. C. ZUNIN, Minerva pediat. **4**:769 (Sept. 15) 1952.

The earliest signs of acute lymphatic leukemia in a 6-year-old boy were fever and abdominal distress. Later there was acute tenderness and spasm in the right lower quadrant. Although the white blood cell count at this time was 3,200 cells, none of them being abnormal, and the red blood cell count was 2,500,000, an appendectomy seemed necessary. The appendix was red and swollen, and several large mesenteric lymph nodes were found. Two days later, the white blood cell count had risen to 26,000, and numerous abnormal cells were found on smear. The child died 15 days later. An autopsy report is given.

HIGGINS, Boston.

TREATMENT OF MEDITERRANEAN ANEMIA (COOLEY'S DISEASE) WITH COBALT SALTS. S. VIRDIS, Minerva pediat. **4**:827 (Oct. 15) 1952.

A dose of 20 to 30 mg. of cobalt chloride in a 1% water solution was given to six children with Cooley's anemia daily over a period of 20 to 26 days. In one case there was an increase in the hemoglobin and the red blood cell count after 10 days; this increase was balanced by a drop in the next 10 days. In none of the other cases was there any change in the blood picture following the medication.

HIGGINS, Boston.

TWO CASES OF ACUTE INFECTIOUS LYMPHOCYTOSIS. G. SCALAMOGNA, Minerva pediat. **5**:123 (Feb. 28) 1953.

Two children, brothers 2 and 5 years of age, were taken sick with acute infectious lymphocytosis seven days apart; this fact suggests that this disease has a shorter incubation period than has usually been thought. In the younger child the disease was initiated by convulsive seizures; otherwise, the mild clinical symptoms and the blood count findings (about 40,000 white blood cells with 85% lymphocytes, persisting for about three weeks) were as have been reported in other cases.

HIGGINS, Boston.

CONSTITUTIONAL THROMBOPATHY IN CHILDREN. N. QUATTRIN, M. MARCACCI, and A. CAMERA, Pediatria **60**:545 (Nov.-Dec.) 1952.

Three cases of thrombopathy in children, 3 to 5 years of age, are reported. The abnormality in each case was congenital, and in none hereditary. In two of the three cases, the abnormality involved the blood vessels as well as the platelets themselves. The symptoms were sporadic and did not respond satisfactorily to any type of treatment, even transfusions.

HIGGINS, Boston.

CONGENITAL MALFORMATION OF THE BLOOD VESSELS IN A LIMB. W. GALLUZZI and A. GIANELLI, Pediatria **60**:609 (Nov.-Dec.) 1952.

Congenital malformations of the blood vessels of the leg are the result of adhesions, anatomical malformations, or arteriovenous communication (aneurysm). A case is cited in which vascular abnormality was secondary to an abnormality in the nerve structure.

HIGGINS, Boston.

ULTIMATE RESULTS OF SPLENECTOMY IN COOLEY'S ANEMIA. I. GATTO and F. JACONO, Pediatria **61**:27 (Jan.) 1953.

Three patients, 26, 20, and 19 years old, with Cooley's anemia had had splenectomies seven years previously. Blood and bone marrow studies were made before and three and seven years after the operation; the data are recorded and compared in charts. After seven years, the red blood cell counts and the hemoglobin percentage had become normal, but nucleated red blood cells persisted in the blood smears, and there was considerable hemolysis present. There was marked erythroblastic growth in the bone marrow; this had led in one case to compression on the spinal cord and a spastic paraplegia. The operation had been followed by increased somatic growth and sexual development.

HIGGINS, Boston.

THALASSEMIA MINIMA. I. GATTO and L. VALENTINO, Pediatria **61**:313 (May) 1953.

Hematological studies were made on 25 persons, the parents or siblings of patients with Cooley's anemia. These people were in good health, but carried the trait of that type of anemia. This condition is called thalassemia minima. The examinations afforded a basis for the diagnosis of thalassemia minima. Each patient showed eliptocytes and microcytes in the blood and a low hemoglobin content in the individual red blood cells or a high osmotic resistance in the erythrocytes, or both.

HIGGINS, Boston.

A NEW HEMORRHAGIC DISEASE ANALOGOUS TO HEMOPHILIA. M. AMATO and A. CAMERA, Pediatria **61**:336 (May) 1953.

A boy 4 years old had several severe bleeding episodes over a period of two years; the bleeding stopped following transfusions. Three of his four brothers had died following bleeding episodes; the fourth brother had died as a baby. Three sisters were well. Study of the child's blood revealed a prolonged clotting time and a deficient conversion of prothrombin to thrombin. The platelets were normal qualitatively and quantitatively. There were no deficiencies in thromboplastinogen, prothrombin, or Factors V or VII. The blood calcium and fibrinogen were normal, and the blood anticoagulants were not increased. The addition of normal serum to the patient's blood resulted in the coagulation and prothrombin time becoming normal. These studies indicated that the patient's condition was due to a deficit in the plasma thromboplastin component of Aggeler (*Proc. Soc. Exper. Biol. & Med.* **79**:692, 1952). No similar cases have been reported in the Italian literature.

HIGGINS, Boston.

A CASE OF DEXTROCARDIA WITH A SINGLE VENTRICLE. FERNANDO FALCON GARCIA, Acta pediat. espan. **9**:706-726 (June) 1951.

Garcia presents the case of a child born of a mother who had an infection of undetermined origin from the first month of pregnancy. All agglutinations, such as typhoid, malta fever, etc., were negative. There was no rash present, and the fever, vomiting, and pain in the right side were unrelieved by any antibiotics, sulfonamides, or other drugs. At no time was there any vaginal bleeding. Wassermann and tuberculin tests were also negative. The illness was present throughout pregnancy, with a few weeks' remission.

The child was intensely cyanotic at birth and had a large umbilical hernia and very poor subcutaneous tissue.

Complete details of physical examination at time of birth are given, with x-rays of thorax, etc. Important findings were a dextroposition of the heart and a liver enlarged and on the left side; the stomach was also on the left side.

Electrocardiographic studies at birth and at 10 months were characteristic of a heart with only one functioning ventricle.

Angiocardiograms at 10 months showed on the right (1) shadow of the vena cava superimposed on the shadow of the descending aorta, (2) no evidence of a pulmonary artery, and (3) contrast media which showed full outline of the heart.

The child at 10 months was still cyanotic and showed the development of a child of 4 months.

The author reviews various anomalies of congenital hearts and concludes that this is a case of incomplete rotation of the heart to the right, with one ventricle functioning and possible absence of the pulmonary artery.

He feels that the mother's illness may have had something to do with the anomaly but also points to a family history of much cardiac pathology. The case is not amenable to surgery. Prognosis is poor.

ALLEN, Santa Fe, N. Mex.

A CASE OF WERLHOFF'S PURPURA. MATOS AGUILAR and CASADO DE FRIAS, Acta pediat. españ. **10**:735 (Sept.) 1952.

The author presents a case of thrombopenic purpura in an 8-year-old girl. He gives all the clinical details and the differential diagnosis.

ALLEN, Santa Fe, N. Mex.

ORDINARY RADIOLOGICAL EXAMINATION IN THE DIAGNOSIS OF CONGENITAL CARDIOPATHY IN INFANTS. F. DE VEGA GOICOECHEA, Arch. pediat. **2**:431-439 (Jan.-Feb.) 1952.

With the new surgery on congenital heart anomalies, the ordinary x-ray and fluoroscopic examination assumes new importance. Angiocardiography and catheterization of the heart should not be attempted except in well-established centers with personnel and equipment for these procedures. However, much can be learned from proper x-rays. The outline of radiological findings in infants is given; the significance of form, size, and position of the heart and large vessels in various positions is discussed and diagramatic cuts are shown with explanations. Films are taken in anteroposterior position and in both right and left anterior oblique positions. Normal and abnormal findings in each position are listed.

The peculiarities of the child's heart as compared with that of the adult are noted.

Several pitfalls must be avoided. Enlarged heart may appear temporarily in a child of a diabetic mother. This disappears after a few weeks. Occasionally an enormously enlarged heart may be noted which on post mortem shows no abnormalities of structure or evidence of infection but only the presence of an enormous enlargement. Von Gierke's disease may give this finding.

ALLEN, Santa Fe, N. Mex.

TRICUSPID ATRESIA AND RIGHT VENTRICULAR HYPOPLASIA. GASTON GUFFAU, Rev. chilena pediat. **21**:266 (June) 1950.

Three cases of tricuspid atresia and hypoplastic right ventricle are described. In the first case the diagnosis was established at the age of one month. The patient died at the age of 3 months, and at autopsy there were found an atresia of the right ventricle and of the pulmonary artery, absence of the tricuspid valve, interauricular communication (foramen ovale), and a patent ductus arteriosus.

In the second case, besides the essential findings of this malformation, there existed a persistence of the left superior vena cava, without an anastomosis with the right superior vena cava (McCotter's Type 1), and a right aortic arch.

The patient of the third case is still alive and will be operated on.

The differential diagnosis is studied in detail.

FROM THE AUTHOR'S SUMMARY.

DETERMINATION OF THE BLOOD VOLUME IN INFANTS. OSCAR CORREA and IGNACIO DUQUE, Rev. chilena pediat. **23**:145 (April) 1952.

A study on the determination of total blood volume in infants is reported. The work included studies of 32 infants of various nutritional and clinical conditions. Blood volume values were obtained from Hill's formula, which is based on the transfusion changes in the hemoglobin

concentration of the recipient's blood. Hemoglobin was determined by the colorimeter method with the use of the Evelyn photoelectric colorimeter.

As compared with the expected blood volume values, in all cases very low figures were found. It is of interest that no relationship could be established between blood volume level and nutritional or clinical status of patients.

FROM THE AUTHORS' SUMMARY.

CHRONIC ADHESIVE TUBERCULOUS PERICARDITIS. AGUSTIN CASTELLANOS, RAUL PEREIRAS, and RENE MONTERO, Arch. med. inf. **22**:1 (Jan.-March) 1953.

This is the first report published in Cuba of a case of chronic constrictive pericarditis with final progression to tuberculous polyserositis successfully operated on.

The authors state that after a thorough review of the medical literature, they have not found any other case in which as many means of research were utilized as in theirs.

The accurate diagnosis arrived at in this case is one of the most significant reasons for the success of the surgery performed. Well-known methods of diagnosis were employed, together with the new cavographic technique, which the authors have described for the first time in medical literature. The methods employed were as follows: simple radiography, electrocardiography, radiokymography, venous tension, determination, speed of circulation, anterior pneumomediastinum, superior cavography, and inferior cavography.

All these tests, done both before and after operation, proved the normality of the cardiovascular function, obtained by resection of the pericardium and costal resection by the method of Brauel, done at the same time.

The anterior pneumomediastinum is of importance in the diagnosis of the cardiac accretion. Condorelli, the author of this method, used it in determining if there were any mediastinal adhesions between the pericardium and the mediastinal structures. Not only in this case, but in many others that the authors have observed, they have been able to prove the great value of this method in the study of the conditions of the mediastinum where cardiac accretion exists and in which this pathological sign can only be identified by the use of this method.

Radiokymography has, in the authors' opinion, been a very useful exploratory procedure not only for diagnostic purpose but also to follow the patients' progress after operation. This method has an advantage in that it shows the surgeon's intervention clearly, facilitating an effective and successful operation, as it localizes those places which are most affected by the pericarditis.

Electrocardiography is considered a compulsory investigation in all cardiovascular disturbances. There are a number of typical alterations in constrictive chronic pericarditis readily observed by electrocardiography which practically completely disappear in those cases in which there has been surgical intervention at the optimum time and with adequate technique.

The determination of the venous tension and the speed of circulation and superior and inferior cavographies constitute important exploratory methods. Cavography will indicate the condition of the large venous trunks, showing any deviation, hardening, compression, etc. It is also indicated if there is any supplementary deep circulation and facilitates the understanding of the hypertension of local and general veins, as well as any retardation in the circulation time.

The authors have described Joselevich's syndrome of cardiac retention, making three major classifications: interpericardiac, extrapericardiac, and mediastinal.

The authors have jointly studied the cause of constrictive chronic pericarditis, as well as the pathogenic, symptomatologic, diagnostic, and therapeutic phases of this condition, in order to draw special attention to this interesting syndrome, which may be completely cured or greatly improved by a correct diagnosis made in the early stages of the disease and by correct surgical treatment.

FROM THE AUTHORS' SUMMARY.

CLINICAL DIAGNOSIS OF THE MOST COMMON TYPES OF CONGENITAL HEART DISEASE. GROSSE-BROCKHOFF, Montasschr. Kinderh. **100**:118-127 (April) 1952.

Two forms of cyanosis are found in congenital cardiac lesions: (1) peripheral cyanosis localized mainly to fingers, nose, ears, and outer portions of the lips when exposed to cold temperatures or after heavy exercise; this type is usually seen only in patients with isolated pulmonary stenosis, and (2) classic morbus caeruleus seen in patients with a right-to-left shunt; in this type cyanosis is present also in the conjunctiva, tongue, soft palate, and inner aspects of

the lips in addition to the sites mentioned above. Sudden changes from the picture of peripheral cyanosis to morbus caeruleus are seen in patients with pulmonary stenosis with patent foramen ovale or interauricular septal defect as right auricular pressure rises. The conditions of such patients are contraindications for the Blalock-Taussig operation, but they are candidates for Brock's direct correction of the pulmonary stenosis.

Cyanosis of the lower with either slight or absent cyanosis of the upper part of the body occurs in complete stenosis of the isthmus of the aorta, with a patent ductus arteriosus entering the aorta distal to the atresia. The opposite picture, namely, more intense cyanosis of the upper part of the body as compared with the lower, is characteristic of complete transposition of the great vessels with a patent ductus arteriosus.

Determination of the oxygen saturation of arterial blood is a useful measure of the amount of venous-arterial mixing which is taking place. Normal oxygen saturation of arterial blood is 93%, while in cases of venous-arterial mixing it can be as low as 70%. In cases of suspected venous-arterial shunts which do not show decreased oxygen saturation, exercising the patient may reveal the drop in oxygen saturation. In cases of tetralogy of Fallot with less than 20% drop in oxygen saturation after exercise, the Blalock-Taussig operation is not indicated, as such patients probably have sufficient collateral pulmonary circulation. There is good correlation between degree of polycythemia and arterial oxygen deficit.

The most important congenital cardiac defects with cyanosis are tetralogy of Fallot, truncus communis, tricuspid atresia, pulmonary arteriovenous fistula, and anomalous venous drainage into the heart. Those with latent cyanosis are Eisenmenger's complex, pulmonary stenosis with patent foramen ovale or interauricular septal defect, and Ebstein's disease with patent foramen ovale and infantile coarctation of the aorta. The noncyanotic types are pure pulmonary stenosis, interauricular septal and interventricular septal defects, patent ductus arteriosus, adult type of coarctation of the aorta, Ebstein's disease without patent foramen ovale, and congenital mitral, aortic, and isolated tricuspid stenosis. In this last group peripheral cyanosis may appear.

The occurrence of a distinct and/or split-second pulmonic sound speaks against diagnosis of pulmonary stenosis, such as in a patient suspected of having tetralogy of Fallot, but it is in favor of an associated patent ductus arteriosus or other lesion increasing pulmonary pressure. Presystolic in addition to systolic murmurs may be heard in interauricular septal defects and can, therefore, be confused with mitral stenosis. Continuous systolic-diastolic murmurs may occur in Eisenmenger's complex (diastolic element due to pulmonary insufficiency) and truncus arteriosus (due to aortic insufficiency), as well as in the classic patent ductus arteriosus. If the patent ductus arteriosus is short and/or wide, the diastolic component of the murmurs may be absent, as pulmonary pressure equals intra-aortic during diastole.

The electrocardiograph in pulmonary stenosis and tetralogy of Fallot shows right deviation or strain and high, peaked P waves. Tricuspid atresia is characterized by left axis deviation or strain.

Cardiac catheterization should include fluoroscopic control, blood samples from the various chambers and vessels for oxygen content and saturation, and pressure determinations.

FROM THE AUTHOR'S SUMMARY.

A CONTRIBUTION TO RESEARCH INTO THE BLOOD SERUM PROTEINS. R. STRYCEK, Pediat. listy **6**:107, 1951.

The physical and chemical properties of the blood serum were examined by means of two methods. One of them evaluates the capillary dynamic properties of the serum; the other is based on the colloid chemical reversible flocculation resulting from the reciprocal reaction of the picric acid solution with the blood serum proteins. The author describes in detail his methods and puts forward his results with normal and pathologic blood serum.

FROM THE AUTHOR'S SUMMARY.

THE SIGNIFICANCE OF FETAL HEMOGLOBIN. R. GOSTOV and J. HOMOLKA, Pediat. listy **7**:150, 1952.

The affinity of hemoglobin in the placental blood for oxygen or its resistance to denatured alkalies was studied in 40 cases. No correlation was found between the affinity or the resistance

and the condition of the mother, the course of the pregnancy and delivery, or the clinical condition of the child in the first 10 days. Four pairs of twins were observed, and it was noted that the resistance of the hemoglobin was greater in unlike twins. The opinion is expressed that the properties of fetal hemoglobin are due to a greater or less degree of anoxemia. It is probable that the greater affinity of fetal hemoglobin for oxygen is one of the provisions to overcome the disproportion between the oxygen needs of the organism and the amount available in the uterus and even for a short time after birth.

FROM THE AUTHORS' SUMMARY.

A TYPICAL COURSE IN CASE OF GAUCHER'S DISEASE IN INFANCY. J. BRACHFELDOVA and J. JANELLE, Pediat. listy **7**:224, 1952.

The authors observed a case of Gaucher's disease in a 9-month-old infant, the onset of which was probably at 6 months of age. The diagnosis was confirmed with certainty by the findings of typical cells in the bone marrow and spleen. Splenectomy was undertaken. After a generalized improvement the child died at the age of 2¼ years. The authors are unable to decide whether the prolonged improvement for three-fourths of a year was due to the splenectomy or whether the case was an atypical one and would have run a similar course without splenectomy.

FROM THE AUTHORS' SUMMARY.

PHYSIOLOGICAL CARDIAC MURMURS IN CHILDREN. J. RINGEL, Pediat. listy **8**:80 (June) 1953.

Four hundred forty-one children 6 years of age were examined in the search for the incidence of physiological cardiac murmurs. These murmurs were found in 42.4% of all the children with no significant difference by sex. A relationship between the incidence of the physiological murmurs and some of the somatic features (height, weight, circumference of the chest, body surface, vital capacity of the lungs, pulse rate, systolic and diastolic blood pressure, thoracocardiac index, transverse diameter of chest and heart), were looked for in the statistical analysis of the material. No certain relationship between the investigated signs and the frequency with which physiological murmurs occurred could be proved. Only the transverse diameter of the heart was bigger in children with murmurs than in children with none. However, this greater cardiac diameter was not linked in any way with pathological changes in the myocardium. The validity of the well-known characteristics of these murmurs was confirmed; they are always systologic murmurs, brief in duration, with a vibrating nature and faint audibility. These murmurs were found most frequently at the apex (41%), in the mesocardium (27%), and over the pulmonary area (20%). In view of the fact that the occurrence of physiological murmurs is not dependent on the child's biotype nor on any of the known factors of the external environment, the diagnosis of physiological murmurs continues to rest on the characteristic features of these murmurs and on the indispensable condition of an entirely normal general as well as cardiological finding.

FROM THE AUTHOR'S SUMMARY.

STUDIES ON CARBONIC ANHYDRASE ACTIVITY IN CHILDREN. RAGNAR BERFENSTAM, Acta paediat. **41**:32 (Jan.) 1952.

A short review of the literature on the enzyme carbonic anhydrase is given. Special weight has been laid on earlier clinical work.

An account of the author's method, which is a modification of the "boat method" of Meldrum and Roughton, is given.

A number of normal values for the carbonic anhydrase activity in the blood of adults, children in various age groups, and infants are given. It is concluded that the activity for newborn infants is about one-third that of adults and that the enzyme activity in infants and children increases with increasing age.

The enzyme activity in the blood of premature infants is shown to be only about 5 to 10% that of adults. The activity increases slowly during the first months of life.

The relationships between the enzyme activity and the degree of maturity as well as length and weight at birth are discussed. There seems to be a good correlation between enzyme activity and maturity.

The clinical importance of the low enzyme activity for cyanosis and vitality in premature infants is discussed. The author has not been able to verify the findings of Stevenson that cyanosis often is associated with low carbonic anhydrase activity.

A parallel relationship between the enzyme activity and the zinc content of the blood is demonstrated.

FROM THE AUTHOR'S SUMMARY.

STUDIES ON THE RESISTANCE OF HÆMOGLOBIN TO ALKALI. LEDA ZANNOS, Acta pædiat. **42**:305 (July) 1953.

Using Singer's test of hemogloblin alkali denaturation, the author examined 61 nonanemic infants aged between 5 days and 2 years and found a fetal type of hemoglobin up to the 19th month of age. There were 12 patients with Cooley's anemia. The values of resistant hemoglobin were above 50% in patients who had not received a transfusion previously. These values were much higher than those found by Singer in similar cases. Thirteen patients had sickle-cell anemia. The values of resistant hemoglobin fell between 5 and 19.5%, coinciding with those found in Negro patients. There were two patients with kala-azar. The hemoglobin was found to be of the normal type.

FROM THE AUTHOR'S SUMMARY.

RETICULOENDOTHELIOSIS MALIGNA. G. M. C. ESKELUND, Acta pædiat. **42**:350 (July) 1953.

After a short review of the present problems in the interpretation of three types of reticulosis, Hand-Schüller-Christian disease, Letterer-Siwe disease, and eosinophilic granuloma, the author describes one case. This he regards as a transitional case between Hand-Schüller-Christian disease and Letterer-Siwe disease. The author discusses the possibility of a biologic progress, starting primarily from an eosinophilic granuloma.

FROM THE AUTHOR'S SUMMARY.

CUTANEOUS FORM OF RETICULOSIS WITH VARIOUS LOCALIZATIONS. G. CARPENTIER and M. RABAEY, Maandschr. kindergeneesk. **20**:177 (July) 1952.

This is a report of a case of reticulosis in a 9-month-old infant, starting with subcutaneous nodules of reticuloendothelial proliferation and localization in the eyes. After four months of partial remission, the infant died of acute hemorrhagic sepsis accompanied by a monocytic leukemia (Schilling type).

The different localizations are discussed. The variability of the clinical picture of reticulosis is stressed.

FROM THE AUTHORS' SUMMARY.

Diseases of Nose, Throat, and Ear

HEARING IMPAIRMENT IN CHILDREN. H. W. KOHLMOOS, California Med. **78**:33 (Jan.) 1953.

Abnormal behavior in children may frequently be caused by impairment of hearing. Early detection of the impairment and of the cause are of utmost importance, not only to prevent irreversible changes in cases in which that is possible but also to permit early beginning of special training for children who are permanently deaf.

Recent studies have shown that deafness of infants may follow rubella in the mother in early pregnancy or kernicterus caused by Rh incompatibilities. Measures to control these disorders are being investigated. Adequate and careful treatment of diseases of the nose, as well as surgical drainage of infected ears when necessary, are important factors in the prevention of hearing loss in children.

PRYOR, Redwood City, Calif.

MANAGEMENT OF OTITIS MEDIA: ANTIBIOTIC THERAPY VERSUS SURGICAL PRINCIPLES. J. W. MCLAURIN, J. Louisiana M. Soc. **105**:262 (July) 1953.

The author calls attention to the frequency of complications following indiscriminate antibiotic therapy of otitis media. The importance and efficacy of myringotomy is no longer widely understood. The author pleads for more rational use of antibiotics and more frequent use of simple surgical principles.

MCGUIGAN, Evanston, Ill.

HERPANGINA: CLINICAL AND LABORATORY ASPECTS OF AN OUTBREAK CAUSED BY GROUP A COXSACKIE VIRUSES. LILLIAN P. KRAVIS, KLAUS HUMMELER, M. MICHAEL SIGEL, and HAROLD I. LECKS, Pediatrics **11**:113 (Feb.) 1953.

The causal relationship between certain Group A Coxsackie viruses and the disease entity herpangina, first reported by Huebner and his associates in 1951, is confirmed.

Outbreaks of herpangina in Philadelphia during the summer of 1951 are described.

Group A Coxsackie viruses of three different immunologic types were isolated from stools or throat swabs or both obtained from 17 out of the 21 patients involved in the outbreaks.

A rise in complement-fixing and neutralizing antibody titers against homologous virus strains during convalescence was demonstrated in three patients.

Asymptomatic carriers of Group A Coxsackie viruses are described. Evidence is presented suggesting that two children with herpangina acquired their infection from such a carrier.

The criteria used in making a laboratory diagnosis of infection with Coxsackie viruses are discussed.

FROM THE AUTHORS' SUMMARY.

COCHLEAR VESTIBULAR SYMPTOMOLOGY OF ACOUSTIC NEUROMA. H. GIROIRE, A. CHARBONNELL, and J. COLAS, Rev. oto-neuro-opht. **24**:306 (July) 1952.

The authors report the cochlear and vestibular changes in eight verified cases of acoustic neuroma. In all cases there was deafness on the side of the tumor and a complaint of unsteadiness. Deafness in all cases was of long duration—2 to 12 years. The unsteadiness in all the cases was more recent—a few weeks to a few months. Tinnitus was noted only in two cases; in one it was bilateral and was present for only a few weeks in a patient who had been deaf for 12 years. Dizziness, which was recent in all cases, was reported in four cases. True vertigo, during which external objects rotated about the patient, was not present. Dizziness was mainly in the nature of unsteadiness with change of position, sometimes accompanied by sudden weakness of the lower limbs. There was a long first stage, of many years' duration, in which the only complaint was unilateral deafness, with no dizziness or buzzing in the ears. The second stage was of more recent onset and shorter duration. Unsteadiness was in the foreground of the clinical picture.

The deafness on the side of the tumor was complete in seven of the eight cases. That the impairment was of the inner ear type in all cases was readily demonstrable by the Weber and Rinne test and confirmed by audiometric investigation. Nystagmus was present in seven cases and was always bilateral, being occasionally more pronounced in one direction. In four cases it was horizontal, and in three, rotatory. In two of the three cases the nystagmus was horizontal in one direction and rotatory in the opposite direction. In one case the unsteadiness was so great that the Romberg sign counld not be tested for. In some cases, in spite of unsteadiness of gait, there was no swaying during the Romberg test. In six cases there was some veering toward one side, sometimes with some degree of retropulsion. Deviation of the outstretched upper limbs was found five times. The authors also note that the vestibular syndrome was usually incomplete. In the second case there was no deviation of the outstretched upper limbs; in the third there was no veering in spite of definite nystagmus, and in the fifth there was no nystagmus but definite veering occurred. In the sixth case there was no deviation of the outstretched upper limbs in spite of bilateral nystagmus and severe unsteadiness. In the first case there was also a so-called disharmonious vestibular syndrome—deviation toward the left in the Romberg test and deviation of the right upper limb. These findings point to a probable central origin for the vestibular disturbances.

In each case there was impairment of the caloric response on the involved side; in two cases this caloric inexcitability was incomplete.

All the tumors removed were relatively large, being about the size of a pigeon egg. They all extended beyond the internal auditory meatus and compressed the brain stem. The deafness and caloric inexcitability are manifestations of peripheral compression of the eighth nerve, and the central vestibular syndrome is due to impingement on the brain stem.

N. SAVITSKY, New York [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

PERITONSILLAR ABSCESS IN CHILHOOD. L. BABLIK, Wien. klin. Wchnschr. **103**:628 (July 31) 1953.

Serial sections of tonsils removed from children and adults were examined to explain the relative rarity of peritonsillar abscesses in children. In children a sharp border exists between muscle, the connective tissue, and the capsule of the tonsil. This border is often not distinct in adults. Between the capsule and the palatine arch extends a fold of mucous membrane which rarely persists in adults. Furthermore, the capsule of the tonsils is thicker and denser in children than later in life, and extracapsular lymphatic tissue is present more rarely.

AMBERG, Rochester, Minn.

Diseases of Lungs, Pleura, and Mediastinum

PROLONGED TREATMENT OF BRONCHIAL ASTHMA WITH CORTISONE. FRANCIS C. LOWELL, IRVING W. SCHILLER, SAMUEL E. LEARD, and WILLIAM FRANKLIN, J. Allergy **24**:112 (March) 1953.

The report describes the experience of the authors in treating 19 asthmatic patients with cortisone continuously or intermittently for a year or more, in the period from December, 1949, to November, 1952.

Although 17 of the 19 patients maintained a satisfactory state of health, no evidence was obtained that the underlying pulmonary disease of which asthma was the manifestation was materially influenced by treatment. In two patients who improved initially, increasing asthma developed under treatment and the results of treatment may probably be regarded as failure. The chief side effects encountered in this group of patients were gain in weight (10 patients) and hypertension (3 patients) although the latter may be coincidental. Recurrent respiratory infections were difficult to recognize and were often associated with relapse or exacerbation of the asthma.

HOYER, Cincinnati.

LOBAR EMPHYSEMA IN INFANTS AND CHILDREN. HARRY W. FISCHER, WILLIS J. POTTS, and PAUL H. HOLINGER, J. Pediat. **41**:403 (Oct.) 1952.

An infant or young child, previously well, who suddenly has dyspnea or cyanosis, or both, may have lobar emphysema. Physical signs and findings on x-ray examination are diagnostic. Bronchoscopy and bronchography may be valuable in excluding intraluminal causes of emphysema. In the five reported cases, the emphysema was secondary to bronchial cartilage abnormalities and intrathoracic vascular abnormalities, and in one case the cause was not discovered. The authors discuss treatment and the indications for lobectomy.

MACDONALD, Pittsburgh.

ROENTGEN DIFFERENTIATION OF PULMONARY TENSION DISORDERS IN INFANTS AND CHILDREN. J. A. CAMPBELL and R. A. SILVER, Radiology **61**:161 (July) 1953.

The primary causes of pulmonary tension rarefactions are congenital bronchial malformations and acquired interstitial and pleural cyst-like formations. Tension states develop as a result of a check-valve mechanism between the distended cavity and its air supply.

Serial chest roentgenograms are the most important single aid in the differential diagnosis. Occasionally bronchography and planigraphy help.

In the congenital group, complete surgical removal is the only definitive treatment, while the acquired forms are best treated conservatively. A system of analysis is presented which the authors believe will help separate the surgical from the nonsurgical types.

ANSPACH, Riverside, Ill.

BRONCHO-PNEUMONIA IN CHILDREN. N. FRONGIA and E. COSTA, Ann. ital. pediat. **6**:169 (June) 1953.

The authors describe the histological changes in 121 cases of infantile bronchopneumonia. They believe that the variety of histopathological reactions in the child's lung is often dependent upon the age of the subjects.

FROM THE AUTHORS' SUMMARY.

REPEATED HEMOPTYSIS IN A FOUR-YEAR-OLD CHILD FOLLOWING THE ASPIRATION OF A SPIKE OF OAT. M. VERROTTI, Clin. pediat. **34**:797 (Dec.) 1952.

A 4-year-old girl presented a diagnostic problem because of frequent attacks of coughing and hemoptysis over a period of 20 days. On the 20th day she coughed up a clot of blood containing an ear of oat about 2.5 cm. long. Then the symptoms rapidly subsided.

HIGGINS, Boston.

MEDIASTINAL PLEURISY. L. SPADON and N. ZUCCONI, Minerva pediat. **4**:834 (Oct. 15) 1952.

Among 6,227 children admitted to the Children's Hospital in Trieste in the past four years, there were only eight cases of mediastinal pleurisy. These cases are reviewed in this article. The disease is frenquently silent and is detected only by x-rays, stratigraphy being of especial value in making the diagnosis. Of the eight cases, six were on the right side of the chest. Cough and dyspnea were frequent symptoms. Projectile vomiting, probably due to irritation of the vagus nerve, occurred in one case. The patients with a tuberculous background for the pleurisy did not wholly recover for about one year, while the nontuberculous patients recovered in about three months.

HIGGINS, Boston.

DIAGNOSIS OF ABDOMINAL CONDITIONS IN CHILDREN BY RETRO-PERITONEAL PNEUMOGRAPHY. N. CARRARA and C. MARESI, Pediatria **60**:137 (March-April) 1952.

Retroperitoneal pneumography is recommended as a simple and safe means for roentgenological examination of the abdominal organs. The air or oxygen is injected just anterior to the tip of the coccyx, with the needle tilted backward.

HIGGINS, Boston.

BRONCHIAL CARCINOMA IN THIRTEEN-YEAR-OLD BOY. V. Gustad, Nord. med. **45**:640 (April 25) 1951.

A boy had been ill for two and one-half weeks before admission to the hospital with general symptoms and vomiting. Later he had a cough and fever. Clinical and roentgenological signs were those of a mediastinal tumor. X-ray treatment was given. The disease lasted two months from the onset of symptoms. Postmortem examination showed that the left apical bronchus was the site of a tumor, microscopically consisting of cancer, with tumor infiltration in the glands, liver, and some vertebrae.

WALLGREN, Stockholm, Sweden.

Diseases of Gastrointestinal Tract, Liver, and Peritoneum

ILEAL DUPLICATIONS. MARK P. FLECHER, SAMUEL M. DAY, and F. GORDON KING, Ann. Surg. **135**:555 (April) 1952.

The ileum near the ileocecal valve is the commonest site of occurrence. Pathologically, all duplications possess three entities: (1) Each is contiguous with and strongly adherent to some part of the alimentary tube; (2) each is lined with a mucosa of some part of the gastrointestinal canal, and (3) each has a smooth muscle coat.

Symptoms are usually those of a partial intestinal obstruction due to encroachment of adjacent intestine, but the principal symptom may be pain from distention of the cyst by large amounts of secreted fluid.

Treatment is operative. Resection of the duplication with adjacent bowel is the treatment of choice in a good-risk patient.

BANTIN, Omaha.

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEO-ESOPHAGEAL FISTULA, DUODENAL ATRESIA AND OTHER ANOMALIES: A CASE REPORT. WILLIAM C. MCGARITY, OSLER A. ABBOTT, and LON W. GROVE. Ann. Surg. **135**:566 (April) 1952.

The case of an infant with esophageal atresia coexistent with tracheoesophageal fistula and doudenal atresia as well as other anomalies is reported in detail.

It has been recommended that this complex situation be treated, first, by a laparotomy with gastrostomy and gastroenterostomy and, secondly, by a thoracotomy with closure of the tracheoesophageal fistula and exteriorization of the upper esophageal stump.

Procedures to reconstruct the continuity of the gastrointestinal tract would be done electively after the age of 2 years.

BANTIN, Omaha.

SURGICAL MANAGEMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS AND TRACHEOESOPHAGEAL FISTULA. N. LOGAN LEVEN, RICHARD L. VARCO, BERNARD G. LANNIN, and LYLE A. TONGEN, Ann. Surg. **136**:701 (Oct.) 1952.

Congenital atresia of the esophagus and tracheoesophageal fistula are discussed in detail. The authors point out that the symptoms associated with this anomaly should be recognized at birth. An increased amount of mucus may be noted filling the mouth and pharynx and drooling from the side of the mouth. Spells of cyanosis occur due to aspiration of mucous. Severe choking spells occur after a few swallows when the child is fed. Mucus and feedings are regurgitated through the mouth and nose. The infant rapidly loses weight, aspiration pneumonia develops, and the patient dies if not given the chance of survival now offered by surgery.

Earlier diagnosis and more careful preoperative preparation and postoperative care, as well as improvement in operative technique, have resulted in reduced mortality in these cases.

A series of 103 cases seen at the University of Minnesota Hospitals and at the Children's Hospital in St. Paul since 1939 are reviewed. In 23 patients a multiple-stage operation was carried out, and 13 are living. In 68 patients a primary anastomosis of the esophagus was performed, and 43 patients are living.

BANTIN, Omaha.

BILATERAL CHOANAL ATRESIA IN THE NEWBORN. J. R. MITCHELL, Bull. Tulane M. Fac. **12**:115 (May) 1953.

Choanal atresia of the newborn should be remembered as a cause of dyspnea. It is treated by early surgery.

McGUIGAN, Evanston, Ill.

THE ACUTE INFECTIOUS DIARRHEAS. JOSEPH FELSEN, J. Louisiana M. Soc. **105**:255 (July) 1953.

The relationship of bacillary dysentery to ulcerative colitis is indicated. The author points out the increase of salmonellosis in conjunction with shigellosis, a parallel increase often unrecognized and unreported. More recently described diarrheas are usually due to primary extraenteric infective agents, viral dysentery, epidemic diarrhea, and staphylococcal food poisoning.

McGUIGAN, Evanston, Ill.

CONGENITAL CIRRHOSIS OF THE LIVER ASSOCIATED WITH INFECTIOUS HEPATITIS OF PREGNANCY. LEONARD B. BELLIN and IRVING W. BAILIT, J. Pediat. **40**:60 (Jan.) 1952.

The virus infectious hepatitis can cross the placental barrier. In this instance there was delivered from a mother with infectious hepatitis an infant with portal cirrhosis.

MACDONALD, Pittsburgh.

VIRAL HEPATITIS. SYDNEY S. GELLIS and DAVID YI-YUNG HSIA, New England J. Med. **249**:401 (Sept. 3) 1953.

The authors present a review of the new developments in the field of viral hepatitis since 1948. Infectious and homologous serum hepatitis are both discussed in a concise summarization which should be read in its entirety.

WHEELOCK, Denver.

ACUTE ABDOMINAL EMERGENCIES IN CHILDREN. H. REICHARD KAHLE, New Orleans M. & S. J. **104**:429 (May) 1952.

The author outlines the cardinal principles governing examination of the child with abdominal pain: care to avoid frightening the child, routine rectal examinations, blood cell counts, urinalysis, x-rays, and early consultation with the surgeon. The manifold abdominal conditions occurring in children are discussed under age groups.

Treatment, both surgical and postoperative, is mentioned for each condition, as well as the importance of careful anesthesia and surgical technique. Proper use of whole blood and parenteral fluids and restoration of electrolyte and fluid balance are stressed, as is careful and adequate use of gastric suction, bronchoscopic aspiration, and hemotherapy.

McGuigan, Evanston, Ill.

Epidemic Shigella Gastroenteritis (Bacillary Dysentery) in an Endemic Area: II. Age-Specific Clinical Aspects. Milton Feig, Pediatrics **11**:145 (Feb.) 1953.

The clinical aspects of acute bacillary dysentery as observed in an outbreak in a general population group in a highly endemic area are presented, and age-specific clinical variations are noted.

The onset was abrupt in all cases and in all age groups. The height of the illness in 84% of 106 cases was reached in 24 hours; this was more rapid than in the "classical" form (three to five days).

The presence of blood in the stools was noted in about 25% of all cases and in about 25% of the 0- through 5-year age group, 50% of the 6- through 10-year age group, 37.5% of the 11- through 15-year age group, and 9 to 10% in the groups 16 years of age and over.

The median number of stools was 9 to 10 daily, with no significant difference among the various age groups.

Fever was present in 65% of the children 10 years of age and under (55 cases) and in 35% of the older age groups (40 cases).

Vomiting is more frequent among the younger age groups. Weakness becomes a more frequent complaint with advancing age.

The median duration of illness was five and one-half days in 100 cases. There was no correlation observed between age and duration.

Recurrences are more frequently observed in the youngest age groups—36% of all patients (14) under 1 year of age and 28% of 39 patients between 1 and 2 years of age. About 9% of the groups 26 years of age and over (34 patients) had recurrences, there were practically none in the intervening groups.

No relationship was observed between the tendency toward recurrences in households with multiple cases as compared with those with single cases.

All the fatalities were in children under one year of age. Two deaths occurred in the patients studied, yielding an age-specific death rate of 14.3% for this group. Five other deaths in patients not included in this report are noted, all being under one year of age; two were caused by "infant diarrhea" and three by bronchopneumonia, with "chronic diarrhea" as a contributing cause.

The role of acquired immunity in the modification of bacillary dysentery can be evaluated from studies of attack rates and from the clinical course of the disease through age-specific groupings. Investigators should clearly define such basic terms as duration, recurrence, etc., when reporting so that necessary comparisons and/or data summation can be made.

From the Author's Summary.

Hepatitis in Childhood. J. W. Perry, M. J. Australia **1**:914 (June 27) 1953.

The author has brought up the difficulty of determining the cause of liver disease in childhood by means of histological studies. Five cases are described in which two patients had severe infectious hepatitis with typical histological findings and evidence of hepatitis probably of intrauterine origin and two had galactose intolerance with histological findings suggestive of chronic hepatitis.

McGuigan, Evanston, Ill.

A Review of Recent Literature on Chemo-Therapy of Enteric Fever. A. K. Dey, Indian J. Pediat. **20**:75 (March) 1953.

A survey of current literature shows chloramphenicol (Chloromycetin) to be the most effective drug for therapy of enteric fever.

McGuigan, Evanston, Ill.

THE AETIOLOGY OF SUMMER DIARRHOEA. E. KAHN, South African M. J. **27**:624 (July 25) 1953.

The author reviews the literature on summer diarrhea. He contends that the cause of diarrhea is to be found in the deranged physiological processes caused by malnutrition, rather than in the search for a causitive organism.

McGUIGAN, Evanston, Ill.

THE EXCRETION OF METHIONINE IN CHILDREN IN HEALTH AND DISEASE: WITH NOTE ON THE VALUE OF METHIONINE TOLERANCE TEST AS AN INDEX OF LIVER FUNCTION. A. SHAFIX ABBASY and E. A. EISA, J. Roy. Egyptian M. A. **36**:265 (March 6) 1953.

Forty sick children were examined for 24-hour methionine excretion and the results compared with those in well children of the same age groups.

Abnormally high figures were obtained in patients ill with tuberculosis, pneumonia, and hepatitis with jaundice. Increased excretion was a constant finding in manifest and severe liver dysfunction, but not in mild cases.

In true hepatitis with jaundice, high excretion of a loading dose of methionine follows a curve which can be used as an index of hepatic insufficiency.

McGUIGAN, Evanston, Ill.

AGENESIS OF THE INTRAHEPATIC BILIARY CANALS. F. MOYSON, P. GILLET, and J. RICHARD, Helvet. paediat. acta **8**:281 (Aug.) 1953.

A case of agenesis of the intrahepatic bile ducts is described in an 11½-month-old girl. The cholangiography showed that the path of the extrahepatic bile ducts was normal. It therefore seems to be a question of abnormal development of the primary bile duct buds, this development being independent of that of the liver parenchyma.

FROM THE AUTHORS' SUMMARY.

ICTERUS PRAECOX ET PROLONGATUS CAUSED BY IMMUN-ANTI-P-BODIES. H. KOLBL and P. SPEISER, Helvet. paediat. acta **8**:367 (Aug.) 1953.

In a 3-month-old infant icterus neonatorum returned when the child was weaned at the age of five weeks. Anti-P antibodies acting as immunizing bodies could be demonstrated. There was no Rh incompatility.

FROM THE AUTHORS' SUMMARY.

PROPHYLACTIC ACTION OF D. D. T. AND OCTA-KLOR AGAINST GASTROINTESTINAL DISTURBANCES IN CHILDREN. S. CORBO, Arch. ital. pediat. e puericol. **14**:324, 1951.

The author has followed in the year 1949 the incidence of children's mortality caused by intestinal diseases in 15 communities of the province of Latina, comparing it with that of the remaining 13 communities not treated with chlorophenothane (DDT) or with chlordan (Octa-Klor). In the first area there were 7.9 deaths per thousand live births, against 14.08 for the control area. The data for the year 1949, together with those for the years 1946, 1947, and 1948 previously reported by the author, attest the great prophylactic value of chlorophenothane and of chlordan against gastroenteric diseases of children.

FROM THE AUTHOR'S SUMMARY.

ACUTE APPENDICITIS IN CHILDREN UNDER FIVE YEARS OF AGE. W. GALLUZZI and A. GIANELLI, Minerva pediat. **4**:350 (May 15) 1952.

Data and discussions are presented regarding 100 cases of appendicitis seen in the Pediatric Clinic of Genoa over a period of 10 years. Five of the cases were in children under 1 year of age, 7 in the 1-year-old group, and 21, 22, and 45 in the 2-year-, 3-year-, and 4-year-old groups, respectively. In this same length of time there were 308 cases in children between 5 and 10 years of age. The younger the child, the more diffuse the abdominal pain, the more frequent the symptoms of vomiting and diarrhea, and the higher the temperature. Gangrene and perforation also were found more commonly in the younger age group. The differential diagnosis calls for consideration of pneumonia, pyelitis, mesenteritic adenitis, constipation, gastroenteritis, colitis, ascariasis, ureteral abnormality, primary peritonitis, and pathology of Meckel's diverticulum.

Unless the child was operated upon within 24 hours of the onset of symptoms, the appendix had perforated in two-thirds of the cases. The mortality rate was zero, 12, 27, and 35% in patients operated upon on the first, second, and third days and after the third day, respectively.

HIGGINS, Boston.

A RARE CASE OF MIKULICZ SYNDROME. S. NORDIO, Minerva. pediat. **4**:789 (Sept. 30) 1952.

A 6-year-old girl presented the typical salivary and lacrimal glandular enlargements of the Mikulicz syndrome. She also had enlargement of the liver and spleen and of many lymph nodes. A differential white blood cell count revealed a high percentage of large and small atypical lymphocytes. Bone marrow smears were normal. Chlortetracycline (Aureomycin) was given. The glandular swelling had subsided in the course of 18 days.

HIGGINS, Boston.

Nervous Diseases

PATHOGENESIS OF INFANTILE CEREBRAL PALSY. MARIN RAMOS CONTRERAS, Bol. méd. Hosp. inf. **10**:12 (Feb.) 1953.

Since the pathology of infantile cerebral palsy is caused by an arrest in the development of the motor system of the encephalon or by a lesion of it during intrauterine life or during the first two months of life, there is a close relation between the disease and the embryology of the encephalon.

The main causes of the disease are intracranial hemorrhage, which accounts for 50% of the cases; acquired infections of the newborn, responsible for 30%, and the remaining 20% of cases are caused by trauma of the mother and fetus, infections and intoxications of the mother, Rh factor, congenital vascular malformations, obstetrical trauma, hemorrhagic disease of the newborn, and infections of the newborn.

FROM THE AUTHOR'S SUMMARY.

BACTERIOLOGICAL ANALYSIS OF THE SPINAL FLUIDS FROM ONE HUNDRED NINETY-FIVE MENINGITIC CHILDREN. JORGE OLARTE and CONSTANTINO REYES, Bol. méd. Hosp. inf. **10**:118 (April) 1953.

Reyes reports that from the spinal fluid of 621 children with cases of meningoencephalitis, bacteria were isolated in 195 cases as follows: Mycobacterium tuberculosis in 109, Diplococcus pneumoniae in 33, Hemophilus influenzae in 19, Salmonella in 6, Staphylococcus aureus in 3, Neisseria meningitidis in 2, Pseudomonas aeruginosa in 2, paracolon bacillus in 2, Alcaligenes faecalis in 2, Proteus mirabilis in 2, Staphylococcus albus in 2, Neisseria flava in 1, Streptococcus pyogenes in 1, a viridans Streptococcus in 1, Escherichia coli in 1, and diphtheroids in 1. In eight spinal fluid samples the germs isolated were not completely identified.

In order of frequency, tuberculous meningitis occupied the first place. Among purulent meningitides, the commonest were those caused by D. pneumoniae, H. influenzae, and Salmonella. A relatively large number of cases of meningitis were caused by pyogenic germs and by others of intestinal origin. In five cases, multiple infections were found. The importance of this is pointed out in relation to the specific therapy.

FROM THE AUTHORS' SUMMARY.

A CASE OF CEREBELLAR OTIC ABSCESS. FERNANDO LOPEZ CLARES, RAMON DEL VILLAR, and ALEJANDRO HERNANDEZ CAMARENA, Bol. méd. Hosp. inf. **10**:304 (June) 1953.

The authors report a case of cerebellar otic abscess with clinical manifestations that corresponded to a meningitis. There were no localization symptoms. The spinal fluid showed increased proteins, positive Pandy's test, and increased leucocytes, especially at the expanses of mononuclear leucocytes. The patient recovered after a tympanomastoidectomy with drainage of the abscess was done.

FROM THE AUTHORS' SUMMARY.

CONGENITAL CEREBELLAR ATAXIA IN IDENTICAL TWINS. O. SCHEEL THOMSEN, Nord. med. **48**:1452 (Oct. 17) 1952.

The parents and a 13-year-old sister were healthy. The twins were born July 20, 1946. The neonatal period was normal. The birth weight of one twin was 2,200 gm. and of the other 2,400 gm. Static development was retarded. The children could lift their heads at 1 year of

age and started walking at 4 years of age. They had had no infectious diseases. When examined in 1951, they had alternating convergent strabismus, reduced muscle tone, ataxia, dysdiadochokinesia, exaggerated knee reflexes, and bilateral Babinski reflexes. Their gait was slow and ataxic. Walking was possible only with the support of both hands.

In the discussion, the author excludes congenital atonic diplegia, Friedreich's ataxia, hereditary cerebral ataxia with spasticity, ataxia after acute infection, and ataxia due to cerebellar diseases, abscesses, or tumors. The author believes that the etiology of the disease is a developmental defect. The symptoms were identical in both twins. Clinical examinations, electroencephalography and encephalography, showed normal conditions.

WALLGREN, Stockholm, Sweden.

Psychology and Psychiatry

PSYCHOANALYTIC ASPECTS OF DISCIPLINE. MELITTA SPERLING, Nerv. Child. **9**:174 (March) 1951.

This is one of several articles appearing in this journal on discipline of children. It is written by one of the outstanding child analysts in the country. She sets forth in this article the basic concepts of discipline as related to child guidance in terms of psychoanalytic dynamic theory. This presentation is particularly timely in that it corrects many popular misconceptions of analytical and psychiatric indulgence, license, and permissiveness.

She points out that true discipline requires that instinctual energies be put at the service of the person so that he is not afraid to use his impulses and is independent of the pressures of the environment in controlling his impulses. The ultimate goal is that he himself is the master of his impulses, neither fearing them nor using them self-destructively or antisocially. She states that this by no means implies an exaggerated permissiveness of so-called progressive schools, nurseries and self-demand systems for infants.

An important goal for parents in our society is to enable the child to tolerate certain amounts of frustration and control of impulses in order that the child can live in a group and conform to required standards. She succinctly points out that failure to achieve this essential adjustment in the child is so frequent now, that many analysts have stated that child neuroses are disappearing in favor of severe behavior disorders and child delinquency.

The second portion of this paper is devoted to case reports, illustrating Dr. Sperling's theories concerning discipline.

The dominant theme in this paper is to correct the popular opinion that permissiveness is the keynote to all analytyical concepts of child-rearing.

WEINFELD, Highland Park, Ill.

NERVOUS FATIGUE IN THE CHILD: THE EXHAUSTED CHILD IN GENERAL PRACTICE. P. P. LYNCH, South African M. J. **27**:489 (June 6) 1953.

Lynch reports that the exhausted child is a very common finding. He blames it partly on too long a day, with too many hours of outside play and too much time spent listening to the radio or watching the movies. Such children sleep badly, with nightmares, sleep-talking, etc. Because the child is frequently active and hard-driving, he is sometimes overworked by ambitious parents.

Poor posture is a common finding, as is poor resistance to infections. These children are at their worst between 4 and 12 years of age. At 8 and 10 years of age the child begins to face competition both in and out of school and, as a result of the continual strain of keeping up, frequently fails to do his best.

The treatment is enforced rest during the day, nonstimulating or nontiring games, and relaxing activities, such as reading and building sets.

MCGUIGAN, Evanston, Ill.

EFFECT OF TERRIFYING EXPERIENCES ON THE BEHAVIOR OF CHILDREN. A. DALLA VOLTA and G. ZECCA, Minerva pediat. **4**:853 (Oct. 31) 1952.

Two children with behavior problems are the subject of a psychological study. A 9-year-old boy had fears centered around a threat of operation if he masturbated and around expulsion from a boarding school following the theft of a small amount of money which he had used to buy

a light to give him reassurance in a dark bedroom. The other child, a 6-year-old epileptic, had night terrors and walked in his sleep. He feared a gardener who had caught him while he and other boys were stealing flowers. Unconscious motivation of behavior following terrifying experiences is discussed.

HIGGINS, Boston.

PSYCHIC DISORDERS AND EPILEPSY IN CHILDHOOD: PSYCHOGENESIS, CHARACTER RETARDATION, GENUINE EPILEPSY. ECKART FÖRSTER, Deutsche med. Wchnschr. **78**:1105 (Aug. 14) 1953.

Attention is called to the lessening percentage of the diagnosis of genuine (i. e., idiopathic) epilepsy in contradistinction to symptomatic epilepsy. Repeated encephalograms were taken in five children. Although they were not free from attacks in between, no essential differences were found. This is not in favor of the contention that a change in the encephalogram can be brought about frequently by an attack itself. Even with the modern technical methods of examination, definite information with regard to the frequency of idiopathic as against symptomatic epilepsy is hardly obtainable.

AMBERG, Rochester, Minn.

GLUTAMIC ACID TREATMENT IN MENTALLY DEFECTIVE CHILDREN. VERA OLDFELDT, Nord. med. **46**:1516 (Oct. 10) 1951.

Sixteen mentally deficient children (I. Q. 42 to 77) and 15 normal children were treated with glutamic acid, 18 gm. per day in three doses for four months. No demonstrable effect of the treatment could be ascertained.

WALLGREN, Stockholm, Sweden.

MONGOLISM AND HEREDITY. SAKARI LAHDENSUU, Nord. med. **46**:1877 (Dec. 19) 1951.

In 61 out of 105 cases of Mongolism, the author found neurological and psychiatric heredity among relatives (feeblemindedness, psychosis, alcoholism in most cases, epilepsy in some, psychopathia, neurasthenia, and congenital developmental defects). Most of these diseases occurred in parents, in sisters of parents, and in siblings. Comparison with a normal group of persons showed that the hereditary factor occurs in a significantly higher degree in families with Mongoloid children than in the general population.

WALLGREN, Stockholm, Sweden.

Diseases of Genitourinary Tract

OBSTRUCTIVE URINARY TRACT LESIONS IN CHILDREN. EDGAR BURNS, California Med. **79**:207 (Sept.) 1953.

Diseases that cause obstruction to the passage of urine in children almost always are caused by developmental defects. The most common site of obstruction in children, both boys and girls is the neck of the bladder.

The importance of recognizing these conditions in the early stage of their development is emphasized by the fact that renal damage that may lead to failure occurs much more rapidly in children than in adults.

Symptoms are often insignificant. Recurrent or chronic infection in the urine should always be regarded as a warning sign. Investigation of the urinary tract in children is relatively easy and with the improved methods of treatment now available cure can be effected in virtually all cases in which irreversible changes in the kidneys have not occurred at the time of recognition.

PRYOR, Redwood City, Calif.

STENOSIS OF THE URETHRAL MEATUS IN BOYS: SURGICAL CORRECTION. JOSEPH H. KAPLAN AND WALTER M. TASEM, California Med. **79**:231 (Sept.) 1953.

Stenosis of the external urethral meatus in boys is not an uncommon developmental defect. It may cause disease higher in the urinary tract and unless corrected may lead to permanent damage. The most effective treatment is meatotomy, which can be carried out in a physician's office by a method described.

PRYOR, Redwood City, Calif.

TWELVE CASES OF TESTICULAR TUMORS OCCURRING DURING INFANCY AND CHILDHOOD. CARL RUSCHE, J. Pediat. **40**:192 (Feb.) 1952.

This report includes eight children with adult teratoma, three with embryonal carcinoma, and one with a dermoid cyst of the testicle. None of these tumors occurred in an ectopic testicle. Rusche says that preoperative irradiation of malignant testicular tumors is senseless. Immediate extirpation is mandatory. Results are that 10 of his 12 patients are alive without evidence of disease; 8 of these children have lived five years or longer and may be considered cured.

MACDONALD, Pittsburgh.

MEIGS' SYNDROME: A REPORT OF A CASE IN A CHILD. WILLIAM E. KNAUS, JOSEPH CAMPOS, and WILLIAM ROSE, J. Pediat. **43**:88 (July) 1953.

Meigs' syndrome is characterized by ascites, hydrothorax, and ovarian tumor. The authors report a case in a 9-year-old girl. The tumor was diagnosed as a functional granulosa- and theca-cell tumor of the ovary. After removal of the tumor, effusions from the abdominal and chest cavities cleared.

MACDONALD, Pittsburgh.

FUNCTIONAL DISORDERS OF MENSTRUATION IN ADOLESCENSE. JANET W. MCARTHUR, New England J. Med. **249**:361 (Aug. 27) 1953.

The author reviews the physiology of menstruation as it applies to the functional disorders of menstruation in adolescent girls. The menstrual and reproductive performance of the adolescent girl is described and followed by an analysis of the factors involved in sexual maturation, i. e., pituitary activation, tissue responsiveness to hormonal stimulation, and integration of the entire process.

Animal experiments and observations are elucidated and correlated with the clinical disorders of menstruation in the young human girl. This report obviously should be part of the knowledge of all pediatricians.

WHEELOCK, Denver.

RADIOGRAPHIC DIAGNOSIS OF HYDROCOLPOS IN INFANTS. LOUIS L. KLOSTERMYER and JOHN J. THOMPSON, Radiology **58**:100, 1952.

An abdominal mass simulating a tumor mass on roentgenograms of the genitourinary tract may be due to imperforate hyman or atresia of the vagina with distention of the vaginal canal (hydrocolpos). Details are given of this condition in a 2-month-old child. Relief of symptoms and disappearance of the mass were obtained by incising the protruding hymen. Three ounces of gray-colored fluid was obtained.

ANSPACH, Riverside, Ill.

KIDNEY FUNCTION DISTURBANCE IN CYSTINOSIS. H. WEBER, Helvet. paediat. acta **8**:348 (Aug.) 1953.

A case of cystinosis in a 1½-year-old child was investigated over a period of five months. The essential characteristics of the electrolyte metabolism were observed. The clinical picture corresponded to the cases previously described, but the metabolic processes were essentially different from those reported by other authors.

Up to now, with the exception of one case, the observed disturbances of renal function seemed to be limited to the proximal tubule. In this case the author had clear evidence of a dysfunction in the distal tubule with inability to elaborate an acid urine, causing a hyperchloremic acidosis. This patient showed a picture of renal acidosis superimposed on the picture of cystinosis. The author observed a disturbance in potassium metabolism causing K depletion. This disturbance could be explained by an inhibition of the carbanhydrase in the distal tubule. As the condition deteriorated, a progressive renal failure developed, causing K retention and death by heart block. The inhibition of carbanhydrase was not limited only to the kidney but seemed to be generalized, because the author found no acid in the gastric juice. The underlying cause of cystinosis is thought to be a lack of phosphatase in the kidney. In this case evidence of lack of another enzyme, carbanhydrase, was obtained.

FROM THE AUTHOR'S SUMMARY.

FAVORABLE RESULTS OBTAINED IN THE TREATMENT OF CHILDREN WITH ACUTE HEMORRHAGIC GLOMERULONEPHRITIS WITH AUREOMYCIN. R. PACHIOLI and E. CHELI, Clin. pediat. **35**:227 (April) 1953.

Seven children with acute hemorrhagic glomerulonephritis responded to chlortetracycline (Aureomycin) therapy by a rapid clearing up of the clinical and urinary findings. Several of the children had previously received penicillin injections without any improvement in the urinary findings.

HIGGINS, Boston.

APPARATUS FOR THE COLLECTION OF URINE OF SMALL BOYS. B. CARLETTI, Minerva pediat. **4**:787 (Sept. 30) 1952.

The apparatus consisted of a rubber adapter which fitted over the penis and was held in place by two straps which passed over the shoulders.

HIGGINS, Boston.

SELECTIVE TESTS FOR RENAL FUNCTION IN ACUTE GLOMERULONEPHRITIS OF CHILDREN. P. DURAND and R. BRUNI, Minerva pediat. **4**:793 (Sept. 30) 1952.

After a detailed discussion of the physiology of the glomerular and tubular function of the kidney, the result of tests of kidney function in 11 children with acute glomerulonephritis is given. In the early stages of the disease there was found a marked reduction in glomerular filtration and some diminution in renal plasma flow and in maximal tubular excretion. As recovery set in, there was an increase in glomerular filtration and renal plasma flow, but the tubular excretion continued to be low.

HIGGINS, Boston.

SODIUM THIOSULFATE CLEARANCE IN INFANTS. L. PARENZAN and F. SERENI, Minerva pediat. **4**:1063 (Dec. 31) 1952.

Clearance tests with sodium thiosulfate and mannitol were done simultaneously on eight normal and three dystrophic infants. The results with each substance agreed very closely. Hence the conclusion is drawn that the sodium thiosulfate clearance test affords a satisfactory measure of glomerular filtration.

HIGGINS, Boston.

OVARIAN TERATOMA IN INFANCY. A. CALAHORRA, Arch. pediat. **3**:647 (March-April) 1953.

Because teratoma of the ovary is a very rare condition, the author presents this case of a girl 18 months old. The illness was at first confused with an acute infection and treated as such. Later a palpable mass and continued pain led to surgical intervention, when a teratoma the size of an orange was removed. Pathological section showed osseous, nerve, and cartilaginous tissue.

ALLEN, Santa Fe, N. Mex.

MACROGENITOSOMIA CAUSED BY INTERSTITIAL CELL TUMOR OF THE TESTIS: A CASE IN A SEVEN AND A HALF YEAR OLD BOY. E. THAMDRUP, Acta pædiat. **42**:369 (July) 1953.

This case is the thirteenth that has been published. The symptoms (premature somatic and genital development) commenced when the patient was 7 years of age. Six months later a tumor of the right testis, measuring 2 by 2 by 3 mm. was removed and on microscopic examination proved to be a typical interstitial cell tumor without signs of malignancy. Following the operation the patient's abnormally rapid development ceased. Ten months later the patient's condition was unchanged, but 18 months after the operation there was a slight regression of the symptoms. After the operation, the remaining testis developed to more than twice its original size, which was considered a compensatory hypertrophy, as there was no palpable tumor and the excretion of 17-ketosteroids, which before the operation was 40.1 mg. in 24 hours, immediately fell to normal values and remained normal. The possibility of malignancy, the result of the hormone analyses, and the regression of the symptoms after operation are discussed. The presence of malignant tumors has been reported only in adults. In addition to the increased excretion of 17-ketosteroids, there was a slightly elevated excretion of estrogenic hormone. The latter is supposed to have been caused by a production in the tumor cells of abnormal steroids

with estrogen effect, or by an excess of normal estrogenic hormone. Regression of the symptoms have not been observed in all the cases reported. The possibility of regression is greater the earlier the patient is operated on after the onset of the symptoms.

FROM THE AUTHOR'S SUMMARY.

PERSISTING BACTERIURIA IN CHILDREN. TORSTEN IVE, Nord. med. **46**:1858 (Dec. 12) 1951.

The author reports a follow-up study of 20 children with persisting bacteriuria after treatment. Two of them had anomalies of the urinary tract, and one had paralysis of the legs and chronic pyelonephritis. The remaining 17 children were followed for one to four years. Three of them still displayed bacteriuria (Escherichia coli). One of these three children was found to have insufficiency of one urethral orifice; one was cured after further treatment.

WALLGREN, Stockholm, Sweden.

Diseases of Ductless Glands; Endocrinology

EFFECTS OF PROLONGED PERCUTANEOUS ADMINISTRATION OF METHYL TESTOSTERONE AND ESTRADIOL ON GROWING MALE RATS. AMOS E. LIGHT and JOHN A. TORNABEN, J. Nutrition **49**:51 (Jan. 10) 1953.

From the results presented by Light and Tornaben, it appears that estrogen treatment of growing male rats induces a period of extreme energy expenditure which causes an increase in appetite for food and water but permits only subnormal growth. Since wound healing is delayed, this may suggest that the hormonal action is mediated through the pituitary and the adrenals (increased weights of these two glands). Decided variabilities in food and water consumption were also obtained in estrogen-treated animals following temperature changes. When the temperature was increased, the quantity of food eaten was reduced much more in the case of the estrogen-treated animals; likewise a larger intake was obtained with lowered temperatures.

The inunction of methyltestosterone was associated with enlargement of the kidneys and possibly increased deposition of protein tissue; also wound healing time was shortened.

In general, it may be stated that the tissues taken from the androgen-treated animals presented a more normal histological picture, whereas estrogen application caused definite changes in many parts of the body.

FREDEEN, Kansas City, Mo.

VALUE OF MEASUREMENTS OF THYROID UPTAKE AND URINARY EXCRETION OF I^{131} IN ASSESSING THYROID FUNCTION OF NORMAL AND CONGENITALLY HYPOTHYROID CHILDREN. WILLIAM A. REILLY and DINA I. BAYER, J. Pediat. **40**:714 (June) 1952.

With radioactive iodine, Reilly and Bayer show that untreated cretins poorly utilize available iodine. The unused iodine is excreted in the urine.

MACDONALD, Pittsburgh.

SPORADIC CRETINISM IN ONE OF FRATERNAL TWINS. THEODORE JAMES, South African M. J. **27**:562 (July 4) 1953.

James reports a case of cretinism manifest in only one of a pair of binovulvar twins. Only four comparable cases are present in the literature. The author suggests that the fundamental reason for sporadic cretinism in this case might be either a genetic deficiency or lacking or insufficient inductive stimulus for the differentiation of the gland from its substrate.

MCGUIGAN, Evanston, Ill.

A CASE OF LAURENCE-MOON-BARDET-BIEDL SYNDROME. P. SALOMONE, Minerva pediat. **4**:761 (Sept. 15) 1952.

An extensive clinical study is presented of a 6-year-old boy with adiposogenital dystrophy, polydactylism, mental deficiency, myopia, and retinal pigment disturbances; these are the characteristic findings in the Laurence-Moon-Bardet-Biedl syndrome. This syndrome is believed to be due to an hereditary and degenerative process in the embryo; pathological alterations of the primitive cells of the diencephalohypophysial cells will explain the various clinical findings.

HIGGINS, Boston.

INDIVIDUAL VARIATIONS IN BODILY DEVELOPMENT AS AFFECTED BY THE ENDOCRINE GLANDS AND THE LAWS REGULATING GROWTH. M. BARBÀRA, Minerva pediat. **4**:1053 (Dec. 31) 1952.

Diagrams are presented to illustrate the various factors which contribute to the deviation of an individual's growth from what may be called the mean. Such factors are endocranial and vegetative, and much of the deviation may be traced to the endocrine glands.

HIGGINS, Boston.

RECURRENCE OF THYMIC HYPERTROPHY AFTER IRRADIATION. E. COMOLLI, Pediatria **60**:571 (Nov.-Dec.) 1952.

Recurrence of the symptoms of thymic asthma and of enlarged thymic x-ray shadows occurred in the cases of two infants, 1 to 3 months of age, after radiation therapy. The shadows receded after further x-ray treatment.

HIGGINS, Boston.

URINARY ELIMINATION OF THE 17-KETOSTEROIDS IN THE FIRST TWO YEARS OF LIFE. G. MURANO and P. DE ANGELIS, Pediatria **61**:40 (Jan.-Feb.) 1953.

The mean daily excretion of the 17-ketosteroids in the urine of newborn infants on the first day of life was 0.6 mg. The amount gradually decreased to 0.075 mg. by the seventh day. Thereafter there was a gradually increased excretion, reaching 0.180 mg. at 6 months, 0.260 mg. at 1 year, and 0.450 mg. at 2 years. The amount excreted can be regarded as an index of the activity of the cells of the hypophysis and the adrenal cortex. The relatively high excretion in the newborn is ascribed to the effect of the mother's hormones.

HIGGINS, Boston.

PROGNOSIS OF DIABETES IN CHILDREN. A. M. H. VOGELENZANG, Maandschr. kindergeneesk. **20**:205 (July) 1952.

The prognosis of diabetes in children is discussed in the light of extensive American statistics. The span of life after the beginning of the disease is lengthened mainly through the use of insulin. However, there is great morbidity and mortality in later years from vascular disease, of which the etiology is still not clear. In well-controlled cases there seems to be less chance of this. The difficulties encountered in comparing different statistics are discussed.

FROM THE AUTHOR'S SUMMARY.

A CASE OF PRIMARY MYXOEDEMA IN WHICH RADIOACTIVE IODINE (I^{131}) WAS EMPLOYED FOR DIFFERENTIAL DIAGNOSIS. J. J. VAN HAEGENBORGH and F. C. KOOYMAN, Maandschr. kindergeneesk. **21**:301 (Sept.) 1953.

The authors report a case of a child 6 years of age with symptoms of hypothyroidism in which radioactive iodine was used in the differential diagnosis between primary and secondary myxedema. Because the patient had been treated unsuccessfully with thyroid extract and because of the peculiar physical signs and the enlarged sella turcica, the authors supposed that there was a hypophyseal component. They did not see any increased absorption of I^{131} in the thyroid gland after prolonged administration of thyrotrophic hormone, and the secretion type remained typical of myxedema. The basal metabolic rate remained the same and the protein-bound iodine did not increase in the serum. On these grounds, primary myxedema was diagnosed, and treatment with 100 mg. Thyranon daily led to marked improvement. The necessity for adequate treatment of hypothyroidism is pointed out.

FROM THE AUTHORS' SUMMARY.

Diseases of Muscles, Bones, Joints, and Lymph Nodes

CONGENITAL DISLOCATION OF THE HIP: TECHNIC OF OPEN REDUCTION. M. BECKETT HOWORTH, Ann. Surg. **135**:508 (April) 1952.

Congenital dislocation or subluxation of the hip can nearly always be reduced and the reduction maintained by a closed method if the diagnosis is made and proper treatment instituted in the first few months of life. Often, after the child has begun to walk, it may not be possible to reduce the hip by manipulation or to maintain reduction.

The author limits his well-illustrated paper to the technique of open reduction based on experience with 300 such operations.

BANTIN, Omaha.

CONSERVATIVE SURGICAL TREATMENT OF MASSIVE CYSTIC LYMPHANGIOMA: WITH THE REPORT OF EIGHT CASES. G. E. FREEMAN, Ann. Surg. **137**:12 (Jan.) 1953.

Cystic lymphangioma most commonly occurs in childhood and is occasionally a pressing problem early in infancy.

Eight cases of cervical, axillary, or cervicoaxillary cystic lymphangioma are reported, one of them in detail and well illustrated. All but one of these occurred in infants or younger children.

With the aid of good anesthesia and supportive measures, it is possible to perform the dissection necessary for removal of such lesions. Cure is easier if radiotherapy or sclerosing agents are not used prior to surgery. If cystic cavities must be left behind, as in the mediastinum, the use of sclerosing agents to aid in their obliteration may be advisable

An amputative procedure as the primary form of treatment is rarely if ever justified, even for very extensive lesions.

BANTIN, Omaha.

EXPERIMENTAL PRODUCTION OF ARTHRITIS IN RATS BY HYPOPHYSEAL GROWTH HORMONE. W. O. REINHARDT and CHOH HAO LI, Science **117**:295 (March 20) 1953.

Evidences of arthritis (joint tenderness, swelling, and roentgenographic changes) were noted in a series of female rats treated with pituitary growth hormone. The frequency with which arthritis developed was low, but it could be vastly increased if adrenals and ovaries were removed prior to administration of the hormone.

FORBES, Rochester, N. Y.

UNILATERAL LIMB LENGTHENING ASSOCIATED WITH HAEMATOGENOUS OSTEITIS IN CHILDHOOD. WALLACE M. DENNISON, Arch. Dis. Childhood **27**:54 (Feb.) 1952.

In 100 cases of acute hematogenous osteitis, the disease affected a long bone on 84 occasions. Measurable increase in length of the affected limb occurred in 20 cases.

Limb lengthening occurs in other conditions in which there is an increased blood supply to the part. Examples of such conditions are mentioned.

Unless appropriate action is taken, secondary postural deformities may follow increase in length of one lower limb.

KELLY, Milwaukee.

CONGENITAL ANTERIOR ANGULATION OF THE TIBIA. J. P. BOUND, H. V. L. FINLAY, and F. CLIFFORD ROSE, Arch. Dis. Childhood **27**:179 (April) 1952.

Two cases of congenital bilateral anterior angulation of the tibia, with fully ossified fibulae, are described. In the first case, angulation of other long bones was present.

The more important papers in the literature are reviewed, most cases being described in association with congenital absence of the fibula.

Cases with congenital bowing or even angulation of the bones of the arms and legs may have tibial angulation as the only obvious clinical feature. The etiology of the condition is discussed.

KELLY, Milwaukee.

IRREGULARITIES OF BONE MATURATION IN CONGENITALLY MALFORMED CHILDREN. H. P. G. SECKEL, Ann. pædiat. **181**:147 (Sept.) 1953.

Localized congenital defects of the skeleton are not infrequently associated with delay in osseous maturation. This can best be studied in unilateral cases. Such delay is clearly non-endocrine in nature. Similar bilateral retardations of various skeletal areas are met with in systemic congenital anomalies of the skeleton, such as mongolism, arachnodactyly, certain types of primordial dwarfism, and many others. In the absence of abnormal endocrine function, particularly hypothyroidism, these retardations must be interpreted in terms of the experience made with the localized unilateral defect. They obviously are of nonendocrine cause and share in the "hypoplastic tendency" of the localized defect. Occasionally demonstrable accelerations of skeletal development in the same patient have been explained in terms of a so-called "progeric tendency."

FROM THE AUTHOR'S SUMMARY.

CONGENITAL SCOLIOSIS. J. M. GUYOT, Helvet. paediat. acta **7**:272 (June) 1952.

The author describes a case of severe scoliosis, with eventration, in a newborn infant. A disturbance in the development of several lower dorsal myotomes occurring in the third week of embryonal life might explain the cause of this anomaly.

FROM THE AUTHOR'S SUMMARY.

POLYTOPICAL ENDOCHONDRAL DYSOSTOSIS WITH DYSPROTEINEMIA AND DISTURBED OSTEOBLASTIC ACTIVITY. G. VON MURALT, Helvet. paediat. acta **8**:295 (Aug.) 1953.

The author describes a hitherto unknown type of polytopical endochondral dysostosis. It can be distinguished from the known types by the asymmetry of the epiphyseal lesions, by a dysproteinemia of the inflammatory type, and by a lowered level of alkaline phosphatase in the blood. The bone biopsy showed a dysplastic ossification disorder: deficient bone formation together with deficient differentiation of the bone marrow. Two factors may be recognized in the pathogenesis of the disease: a constitutional factor and an inflammatory factor of as yet unknown cause. The diagnosis is discussed in detail.

FROM THE AUTHOR'S SUMMARY.

ASSOCIATED CONGENITAL MALFORMATIONS: SUBLUXATION OF THE KNEE AND LUXATION OF THE HIP. ANDRE MEGEVAND, Helvet. paediat. acta **8**:371 (Aug.) 1953.

A case is described of congenital dislocation of the knee associated on the same side with a dysplasia coxae luxans. The subluxation of the knee was orthopedically reduced when the patient was 5 days old. A year later the child was reexamined because he limped. Examination revealed a dislocation of the hip. This dislocation was reduced by Lorenz's method.

It seems that in such cases the dislocation of the hip is of a special nature; the mechanical factors due to retraction of the quadriceps play an important role and favour the dislocation of a hip so predisposed.

FROM THE AUTHOR'S SUMMARY.

OBSTETRICAL SEPARATION OF THE LOWER EPIPHYSIS OF THE HUMERUS. CAMERA'S OBSTETRICAL LESION. M. VACIRCA and R. PIZZETTI, Pediatria **60**:649 (Nov.-Dec.) 1952.

Good functional recovery in cases of obstetrical separation of the lower epiphysis of the humerus is obtained only if the diagnosis is made early and treatment instituted immediately. Within the first few hours after birth a manual bloodless reduction is possible. Later a manual osteoclasia is necessary, and in some cases an open reduction.

HIGGINS, Boston.

STEINERT'S DISEASE: SPECIAL ATTENTION TO THE PROBLEM OF CATARACT. E. AZZI, Arq. neuro-psiquiat. **9**:1 (March) 1951.

Siblings with dystrophia myotonica, or Steinert's disease, were studied with other members of their families. One of the patients presented a picture of Thomsen's disease, and 13 years later dystrophia myotonica (myotonia atrophica), with incipient cataract, bilateral ptosis, defective extraocular and mimetic musculature, atrophy of distal muscles with foot drop, and testicular atrophy with impotence, developed. In the second sibling pair, a woman with a typical syndrome had a brother four years older with the same disease. Small cysts were found in her ovaries at operation. In the man the disease was more advanced, with repeated attacks of myocardial insufficiency, due probably to involvement of heart muscle. In the third sibling pair, the younger one had a less advanced form of the disease, with early atrophy of the distal parts of the extremities and definitive myotonia, but no dysendocrinism. There were numerous cases of cataract in the collateral and direct descendants in this family.

Hoffmann was the first to point out (1904) the relation of cataract to this disease. Cataract is more important than other ocular complications because it may be the presenting symptom of the malady and may occur without any of the other characteristic signs. The author states that cataract is present in 100% of the cases of dystrophia myotonia if the slit lamp is used for diagnosis. It is found in members of the families in which dystrophia myotonica is present without other evidence of the disease. Azzi describes the coexistence of the Vogt variety of cataract—multicolored punctiform clouding in the anterior and posterior cortices—and dystrophia

myotonica. It is difficult to identify this cataract with that due to parathyroid dysfunction. The author prefers to consider that cataract in this disease is due to diencephalic dysfunction. The absence of cataract in Thomsen's disease does not stamp it as a separate clinical entity. The later appearance of features of dystrophica myotonica in a case at first considered one of Thomsen's disease is cogent evidence for the latter disease being an earlier variant of the full-blown syndrome.

N. Savitsky, New York [A. M. A. Arch. Neurol. & Psychiat.].

A Case of Renal Osteoporosis with Aminoaciduria. S. Van Creveld and P. Arons, Maandschr. kindergeneesk. **21**:294 (Sept.) 1953.

The authors give a description of further experiences in a case of a boy with a particular form of renal osteoporosis with aminoaciduria, successfully treated with dihydrotachysterol.

From the Authors' Summary.

Diseases of Eye

ACTH and Cortone in Eye Diseases. Richard C. Gamble and Walter Segall, Illinois M. J. **103**:101 (Feb.) 1953.

Cortisone used locally is extremely effective in blocking inflammation of the anterior half of the eye. For inflammations of the posterior segment, cortisone (Cortone) or corticotropin (ACTH) given internally should be used. Further study is needed to evaluate the effect in corneal diseases and in retinitis pigmentosa.

Barbour, Peoria.

Trachoma in the South African Bantu. C. Amies, N. L. Murray, J. Graham Scott, and R. St. H. Warren, South African M. J. **26**:362 (April 26) 1952.

Examination of all children in two native villages showed trachoma in 94%. Sixty-eight of 503 children had normal lids. Of these, 38 were found to have the diagnostic pannus, without corneal scarring. In children under 2, only 5% showed no infection, while 100% of those 3 and 4 years old were infected.

McGuigan, Evanston, Ill.

Retinoblastoma. Feliciano Palomino Dena, Lucina Villegas Leon, Renan Murillo Fajardo, and Sabino Silva Zeron, Bol. méd. hosp. inf. **10**:258 (June) 1953.

Comparisons between the results arrived at by foreign authors and those arrived at by the authors of this article have been made in every chapter of this paper.

The authors analyzed 65 cases of retinoblastoma which were confirmed by the pathologist in 55 cases and followed up in 55 instances, with the following conclusions:

1. As a rule its frequency is the same as that reported by foreign authors.

2. Heredity has not been found in the present series as a contributory factor, though this may be attributed to the short period of observation.

3. Pathological characteristics of retinoblastoma were pointed out in order to clear up the apparent nonconformity of ideas regarding the classification of neuroectodermic tumors of the retina.

4. This disease has very significant clinical signs and symptoms that allow the doctor to establish an accurate diagnosis. This should be worked out as soon as possible, considering the very serious outlook of the disease.

5. In order to reach a diagnosis soon, a thorough examination should always be made.

6. The confusing name of pseudogliomas referring to such entities as could be confused with the retinoblastoma should be abandoned, and the real diagnosis should be sought in every case.

7. The prognosis of this ailment has changed for the better, owing to a better knowledge of the disease, better examination procedures, and better therapeutic weapons.

8. The best treatment nowadays is a combination of surgery and x-ray. Healing can be promised when the tumor is found during the first, or intraocular, phase. During the hypertensive

one, success may be attained, and there is still some hope during the third phase. Surgery alone is only permissible at the first stage of the disease, and it should always be followed by a long and careful follow-up.

9. For the first time we may speak about "prophylactic irradiation" of the opposite eye in order to prevent new tumor growth. It should be emphasized as the special method.

10. As a result of better prognosis and better equipment patients afflicted with this disease may have a better outlook than those with cancer in some other part of the body.

FROM THE AUTHORS' SUMMARY.

Cutaneous Diseases; Allergy

STUDIES IN FOOD SENSITIVITY: II. EFFECT OF PROTEIN DIGESTION ON ANTIGENICITY OF FOODS AS DETERMINED BY SKIN TESTS AND CLINICAL FOOD TRIALS. SAMUEL BLOOM, HARRY MARKOW, and BERNARD REDNER, J. Allergy **24**:64 (Jan.) 1953.

Standardized extracts of cocoa, orange, beef, wheat, milk, egg, chicken, corn, potato, and string bean and pepsin and trypsin digests of these foods were used in this study.

Thirty patients with various allergic manifestations were given skin tests with the extracts and with each of the digests.

Each patient was given a feeding test with each of the foods under study.

The results obtained in the study lead the authors to believe that foods play a minor role as compared with pollens, inhalants, and infection in cases of allergy.

HOYER, Cincinnati.

MERCUHYDRIN SENSIIVITY: REPORT OF A CASE. WILLIAM H. HARRIS JR., J. Allergy, **24**:73 (Jan.) 1953.

Severe systemic symptoms occurred in an adult after the intramuscular injection of meralluride (Mercuhydrin). The reaction followed the fifth dose of meralluride given on the sixth day after the initial injection. Increasingly severer reactions followed the sixth and seventh doses given on the 16th and the 26th days, respectively. The patient recovered subsequently with the use of mersalyl sodium and theophylline (Salyrgan-Theophylline) administered intramuscularly without untoward reaction. A positive skin test was obtained 27 months later to blood serum obtained from a patient receiving meralluride intramuscularly.

Reactions resulting from acquired sensitivity to meralluride have been reported in 11 patients, with one fatality.

HOYER, Cincinnati.

THE SO-CALLED ANGIONEUROTIC EDEMA. EGON BRUNN, J. Allergy **24**:97 (March) 1953.

The chief diagnosis was acute circumscribed edema in 132 patients in attendance at the allergy clinic of the university hospital in Copenhagen, Denmark.

An unquestionable allergic history was obtained in 55% of the group. An allergic cause could be presumed in another 18%. In the remaining 27% no cause could be demonstrated.

Acetylsalicylic acid and barbituric acid compounds was the cause of the edema in 38 of the 73 cases in which there was an allergic history; food allergy was the cause in 18 cases; 11 were due to pollen or contact allergy.

The author believes that acetylsalicylic acid and the barbituric acid compounds should be used cautiously in the treatment of patients with a known allergic background.

The author believes too that there is no reason to designate these patients as neurotics, and that the term "angioneurotic edema" should be discarded. He states "The future may justify the term of "allergic edema," but till then I would prefer a more neutral term (Quincke's edema, circumscribed edema, or angioedema)."

HOYER, Cincinnati.

THE EFFECT OF ANTIHISTAMINICS ON PLASMIN ACTIVITY. RIVA RIPSTEIN SOICHER, D. PATRICIA SMITH, and BRAM ROSE, J. Allergy **24**:106 (March) 1953.

The experiments were designed to study the effect of antihistiminics on an active preparation of bovine plasmin (Loomis's fibrinolysin). The effects were compared to those of two known active plasmin inhibitors, Loomis's antifibrinolysin and pancreatic trypsin inhibitor.

Eight different antihistiminics showed no significant inhibition of plasmin in vitro. Antifibrinolysin, pancreatic trypsin inhibitor, and dilutions of human serum led to marked inhibition of the enzyme.

HOYER, Cincinnati.

PEDIATRIC ALLERGY: A REVIEW OF THE RECENT LITERATURE. VINCENT J. FONTANA, J. Allergy **24**:174 (March) 1953.

A concise review showing the relationship of sensitivities to foods, micro-organisms or their products, chemicals, drugs, or pollens to some abnormal conditions seen in children. Such conditions as bronchial asthma, atopic dermatitis, eczema, urticaria, gastrointestinal disturbances, and migraine were mentioned.

The review includes a group of articles stressing the emotional etiological component which may precipitate the onset of clinical symptoms such as personality changes, hyperirritability, hyperactivity, fatigue, and behavior abnormalities.

There are a group of miscellaneous conditions reported, in which the symptoms were relieved by the elimination of an offending food, organism, or parasite. Such clinical conditions were purpura, disturbances in growth patterns, convulsive disorders, grand mal, petit mal, transient synovitis of the hip joint, eosiniophilia of the blood and bone marrow, and eosinophilic granulomatous lesions of the liver containing Ascaris lumbricoides larvae.

It is necessary to think of the possibility of an allergic state in those children whose symptoms do not respond to conventional treatment, especially if there is a family history of allergy.

HOYER, Cincinnati.

KAPOSI'S VARICELLIFORM ERUPTION. G. ZARBIN, Minerva pediat. **4**:781 (Sept. 30) 1952.

Zarbin reports the cases of eight infants, 4 to 16 months of age, in whom there developed varicelliform eruptions associated with high temperature, prostration, and in most cases gastrointestinal disturbances. Each of the children had eczema; none had been vaccinated; only one of them had been exposed to a person with herpes simplex. Treatment was by chlortetracycline (Aureomycin), penicillin, and sulfonamides. The febrile state lasted about eight days irrespective of when the medication was started. The benefit of the drugs was probably only prophylactic against secondary bacterial infections.

HIGGINS, Boston.

PRINCIPAL ALLERGENS CAUSING ALLERGEN PATHOLOGY INHALED BY CHILDREN LIVING IN BIELLA. A. FORTINA and E. GIOVANNINI, Minerva pediat. **5**:113 (Feb. 28) 1953.

Many skin tests were made on 133 children, most of them having asthma due to the inhalation of an allergen. Seventy-four per cent of the children showed positive skin reactions to pollens, 72% to epidermal tissues or derivatives, and 53% to other allergens. Only 20% of the children reacted to the pollens alone, only 7% to the epidermal tissues and derivatives alone, and only 2% to the other allergens alone. The allergens most frequently causing positive tests were grasses among the pollens, duck and goose feathers among the epidermal derivatives, and dust among the other allergens. These observations were made with children living in northern Italy.

HIGGINS, Boston.

TREATMENT OF TINEA FAVOSA IN CALABRIA. G. CASTORINA, Pediatria **60**:328 (May-June) 1952.

An epidemic of ringworm of the scalp occurred in an orphanage in Calabria, involving 48 children under 10 years of age. Epilation was obtained in 30 children by thallium and in 18 by thallium (one-half dose) and x-rays. The use of thallium alone was the more successful, the epilation being complete in 14 to 20 days. The author recommends the thallium treatment as being practical, economical, and efficient, despite the claims of others that thallium is a dangerous drug on account of its toxicity.

HIGGINS, Boston.

CASE OF WEBER-CHRISTAN SYNDROME. P. POLETTI, Pediatria **61**:95 (Jan.-Feb.) 1953.

The Weber-Christan syndrome may be defined as a nonsuppurating nodular panniculitis, with relapsing fever. The present case is that of a 10-year-old girl who had recurring red nodular swellings on the skin of the calves, thighs, and buttocks, each lasting about 10 days. Biopsy of a lesion placed the pathology in the subcutaneous adipose tissue. Antibiotic therapy was ineffectual. The child's temperature did not go higher than 38.5 C. (101.3 F.).

HIGGINS, Boston.

RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA. C. IMPERATO, Pediatria **61**:389 (May) 1953.

A case is reported of recessive dystrophic epidermolysis bullosa in a boy 17 years old. A sister, who had died at the age of 23 months, had the only similar case revealed in the family history. The first bullae were noted when the boy was only 2 weeks old. The child showed impaired physical and mental development (infantilism). The skin lesions were many and large. The patient had one of the most severe cases of this condition reported in the literature.

HIGGINS, Boston.

Teeth and Dentistry

FLUORINE AND DENTAL CARIES. A. B. P. AMIES and P. PINCUS, M. J. Australia **2**:41 (July 11) 1953.

The author points out the unquestioned value of fluorination of water in reducing the incidence of dental caries, but indicates that at present nothing more than a temporary control can be proved. Studies in England indicate that fluorination causes only a lag of a few years in development of caries. The difficulty in assessing caries incidence statistically is mentioned. No mention is made of studies carried out in this country over the past few years where such control has been accurate.

MCGUIGAN, Evanston, Ill.

TENOPLASTY FOR CORRECTING THE NONRIGID SUPINATION DEFORMITY OF THE FOOT. JUAN FARILL, Bol. méd. Hosp. inf. **10**:123 (April) 1953.

Farill describes a technique for the transfer of the tendon tibialis anticus through a subcutaneous tunnel, in order to be sutured to the distal end of the one of the peroneus brevis while the foot is maintained in pronation. In this way the short tibialis anticus can correct the supination of the foot when the pronation can be hypercorrected passively but not actively, as is frequently seen in treated club foot. The proximal end of the peroneus brevis is then attached to that of the peroneus longus. A similar technique can be performed using the latter when it is deemed necessary. This technique was devised in view of the shortened length of the tibialis anticus tendon on being anchored into the cuboid or the fifth metatarsal bone.

FROM THE AUTHOR'S SUMMARY.

Miscellaneous

THE RELATIONSHIP OF HUMAN PLASMA PROTEINS IN THE ACTION AND TRANSPORT OF PENICILLIN. BERNARD HALPERN, RALPH E. DOLKART, JAMES B. LESH, RUSSELL L. KUTZ, FREDERICK L. DAY, and BERNARD WOLNAK, J. Pharmacol. & Exper. Therap. **103**:202 (Oct.) 1951.

This study was made on two normal human subjects given intramuscular doses of 300,000 and 500,000 units of crystalline penicillin G in saline. Studies were made on blood drawn one hour later. For this summary the essential conclusion is as follows:

It appears that though a protein-penicillin complex occurs it is of a nature or type which is readily dissociated when the penicillin concentration is low. It therefore seems indicated that of its protein interaction.

PILCHER, Cleveland.

THE ABSORPTION OF NICOTINE IN TOBACCO SMOKING. L. A. GREENBURG, DAVID LESTER, and H. W. HAGGARD, J. Pharmacol. & Exper. Therap. **104**:162 (Feb.) 1952.

Studies by other observers on the nicotine content of tobacco smoke have shown markedly differing results. In this study mechanical methods, simulating human tobacco smoking, were used which no doubt gave much more accurate results than methods of examining smoke from human exhalations. Cigarettes and cigars were used, and the nicotine present per gram of dry tobacco averaged 17.2 mg. in cigarettes and 17.4 mg. in cigars, the extremes varying little from these figures. In each experiment, about 0.7 gm. of tobacco was burned, and the butt left from a 7 cm. cigarette was 2 cm. in length. The nicotine in smoke of moist cigars and cigarettes was practically 20% of that in the tobacco and 35 to 37% of that in the dry tobacco. This means that smoke from the dry tobacco contained from 70 to 90% more nicotine than smoke from the moist tobacco that contained 11% water.

The authors concluded that in smoking cigarettes or cigars only a small portion of the nicotine content of the smoke is retained in the body with noninhalation, whereas with inhalation virtually all of the nicotine is absorbed.

Since inhaling is very common among cigarette smokers and rare among cigar smokers, it is likely that the former absorb considerably more nicotine than the latter.

PILCHER, Cleveland.

ABUSE AND MISUSE OF ANTIBIOTICS. WENDELL H. HALL, Minnesota Med. **35**:629 (July) 1952.

The indiscriminate use of antibiotics is condemned. The author warns of the possibility of converting a minor illness into a major disaster through the development of serious side-effects, hypersensitivity states, and alteration of normal flora in the gastrointestinal tract.

The serious hazard of an increase in prevalence of bacteria which are resistant to antibiotics is pointed out.

STOESNER, Minneapolis.

SOME SPECIAL PROBLEMS IN THE USE OF ACTH AND CORTISONE THERAPY IN CHILDREN: A REPORT OF THREE CASES. REGINALD WILSON and RICHARD ROWE, J. Pediat. **40**:164 (Feb.) 1952.

From Vancouver, Wilson and Rowe report the cases of three patients with unusual and unexpected side-effects from corticotropin (ACTH) and cortisone therapy.

A boy who had nephritis and edema was treated with cortisone. He died. Autopsy showed thrombi in the renal veins and the pulmonary artery.

A girl had dermatomyositis. Cortisone gave some temporary syptomatic improvement, but hyperlipemia developed and cortisone therapy was stopped. After six weeks, treatment with corticotropin was started. The patient again improved, but paronychial infection developed, and hyperlipemia reappeared. Corticotropin therapy was stopped. The dermatomyositis was basically unimproved. The hyperlipemia gradually disappeared.

Another girl had alopecia totalis. She was given corticotropin. As a result, her sugar tolerance was impaired. This carbohydrate disturbance was benign and reversible.

MACDONALD, Pittsburgh.

PEDIATRIC RESIDENT PROGRAM IN A NONUNIVERSITY HOSPITAL. H. E. THELANDER, J. Pediat. **40**:224 (Feb.) 1952.

Thelander outlines the many facets of a pediatric resident training program. In justice to the subject, it cannot be reported in abstract.

MACDONALD, Pittsburgh.

GALACTOCELE IN A MALE INFANT. SAMUEL P. BESSMAN and JOHN C. LUCAS, Pediatrics **11**:109 (Feb.) 1953.

A case of a galactocele in a 17-month-old male infant is reported, with identification of the sugar contained in the galactocele as lactose.

A hypothesis for the formation of the infantile form of galactocele is presented.

FROM THE AUTHORS' SUMMARY.

LATE SEQUELAE FOLLOWING CANCERICIDAL IRRADIATION IN CHILDREN: A REPORT OF THREE CASES. WALTER T. MURPHY and DAVID L. BERENS, Radiology **58**:35 (Jan.) 1952.

Case 1 was that of a malignant tumor diagnosed as a Ewings' tumor of the right femur when the child was 4 months of age. Five years after treatment there was marked shortening of the right femur.

Case 2 was that of a 2-year-old girl who had a large nonremovable Wilms's tumor low in the abdomen. (Two attempts to remove the tumor failed.). After roentgen treatments the tumor mass reduced in size and, 11 years later, after several courses of deep therapy, there was marked reduction in the size of the left ilium and distortion of the pelvis. A final diagnosis after reexamination of the earlier biopsy was neuroblastoma.

The third patient was a boy 9 years of age with a squamous-cell carcinoma of the roof of the mouth, and in this case roentgen therapy left marked deformity of the maxilla.

The authors conclude that cancericidal irradiation in children will induce permanent somatic tissue changes, manifested by retardation of bone growth, deformity of muscular bony architecture, atrophy and scarring of skin, and fibrosis of subcutaneous tissues. These changes cannot usually be avoided if a clinical cure is the goal of the radiological procedures.

ANSPACH, Riverside, Ill.

CLINICAL PAPER CHROMATOGRAPHY. F. HERMANN, H. BICKEL, and G. FANCONI, Helvet. pediat. acta **4**:397 (Nov.) 1949.

Paper partition chromatography was introduced in 1946 as a diagnostic method for the qualitative differentiation and quantitative assessment of aminoacid mixtures, reducing substances, etc., in the various body fluids. The technique of one- and two-dimensional paper partition chromatography in a clinical laboratory and the identification of the aminoacid spots are described. Attention is drawn to the possibilities of error inherent in this method.

FROM THE AUTHORS' SUMMARY.

NEOPLASMS IN CHILDREN: CONCEPT OF BENIGN AND MALIGNANT. MAXIMILIANO SALAS M., Bol. méd. Hosp. inf. **10**:249 (June) 1953.

Salas reports that benign tumors represent localized tissue proliferations, strictly circumscribed and encapsuled, of slow growth which takes place by expansion without the production of metastasis or reproduction after excision. These tumors exhibit a high degree of cellular differentiation and keep their structural characteristics throughout their evolution, although frequently regressions are seen. Such growths are not essentially dangerous and usually do not affect the general condition of the body.

On the other hand, malignant tumors are infiltrative proliferations that penetrate into the interstices of neighboring tissues, are not encapsuled, have a fast growth, frequently produce metastasis, and reproduce after excision. Their structural differentiation is imperfect, in varying degrees, and usually there are no regressive alterations. They are essentially dangerous and affect the body as a whole, usually ending fatally.

However, as Willis points out so insistently, benignity and malignancy are relative concepts, and not totally different biological qualities. These two terms are different terms used in pathology because of the prognostic value, but it should be kept in mind that between the two extremes there are intermediate types for which the prognosis of benign or malignant must be qualified.

FROM THE AUTHOR'S SUMMARY.

TOBACCO POISONING WITH SINOAURICULAR BLOCK. AKE GYLLENSWARD and FOLKE NORDBRING, Acta Pædiat. **42**:356 (July) 1953.

The authors describe a case of sinoauricular block due to nicotine poisoning. A short review of the literature on the subject is given. Only three similar cases have been found.

FROM THE AUTHORS' SUMMARY.

GENETICS AND CLINICAL PEDIATRICS. J. A. BÖÖK, Nord. med. **46**:1513 (Oct. 10) 1951.

Adequate risk figures for children of affected persons and for siblings of an affected child are available with respect to an appreciable number of genetic diseases and defects. When such disorders are due to single gene differences and the gene concerned shows complete penetrance, the risk can be calculated according to Mendelian segregation. In most cases, however, gene action and the type of transmission are still imperfectly known. The penetrance may be low, i. e., the gene effect may be considerably altered by environmental influences; for such disorders empiric risk figures can be calculated which are independent of etiologic theories. Information about the purely statistical risk should be useful for the general practitioner as well as for the patient who is worried about the appearance of a genetic disorder in his family. The article is illustrated by two tables with English text, the figures of which represent reasonable estimates only. The incidences are those corresponding to conditions among Scandinavian populations.

WALLGREN, Stockholm, Sweden.

REACTIONS IN TREATMENT WITH PENICILLIN. M. K. POLANO, Maandschr. kindergeneesk. **20**: 194 (July) 1952.

Untoward reactions caused by penicillin are to be divided into Jarisch-Herxheimer reactions and allergic reactions. It is not possible to prevent Jarisch-Herxheimer reactions by diminishing the initial doses of penicillin in congenital syphilis. The allergic reactions may be subdivided into urticaria and dermatitis. Sensitization may be caused by previous penicillin treatment, especially by topical treatment with penicillin. Sensitization also seems to be caused by dermatomycosis.

FROM THE AUTHOR'S SUMMARY.

THE PROBLEM OF EXPERIMENTATION ON HUMAN BEINGS. MICHAEL B. SHIMKIN, OTTO E. GUTTENTAG, ALEXANDER M. KIDD, and W. H. JOHNSON, Science **117**:205 (Feb. 27) 1953.

This is essentially a symposium in which the use of man as an experimental animal is discussed from four points of view: (1) M. B. Shimkin, in discussing the research worker's point of view, ends his discussion by stating that science per se is neither moral nor immoral, it becomes moral or immoral only as moral or immoral human beings use its powerful techniques; (2) Dr. O. E. Guttentag considers the physician's point of view and warns that we must be alert with ourselves lest, in our zeal for truth, we create healthy bodies at the cost of morally dulled minds; (3) A. M. Kidd considers the limits of the right of a person to consent to experimentation on himself and the legal risks of the physician who conducts research on human beings, whether it be solely for the advancement of science or therapeutic trials of new drugs and operative techniques; (4) W. H. Johnson emphasizes the distinction, based upon military necessity, between civilian and military personnel.

This symposium should be read by all who are engaged in clinical "investigation."

FORBES, Rochester, N. Y.

ENHANCEMENT OF LEAD EXCRETION IN HUMANS BY DISODIUM CALCIUM ETHYLENEDIAMINE TETRAACETATE. M. RUBIN, S. GIGNAC, S. P. BESSMAN, and E. L. BELKNAP, Science **117**:659 (June 12) 1953.

This compound, known under the trade name Versene, is a synthetic chelating agent forming strong un-ionized soluble complexes with many divalent and trivalent cations.

For use in human subjects, the inclusion of calcium counteracts the action of the parent compound in producing hypocalcemia.

The drug was administered intravenously to four patients suffering from lead poisoning in daily doses of 0.5 to 1.0 gm. Urinary lead excretion rose markedly over pretreatment levels. No statement is made about the clinical course of the patients.

On the basis of animal observations, this compound, and other chelating agents, may prove to be useful in other types of heavy-metal poisoning.

FORBES, Rochester, N. Y.

"Periodic Fever" and Its Differential Diagnosis. W. H. Hitzig and G. Fanconi, Helvet. paediat. acta **8**:326 (Aug.) 1953.

Seven cases, all in boys, are described. They have in comon periodic short attacks of fever over months or years. An organic ailment which could explain the fever was found in four cases. In one, Hodgkin's disease (with Pel-Ebstein's type of fever), in two others, a kidney disease with fatal outcome (polycystic kidney?), in the fourth, encephalitis was suspected among other things on the basis of the findings on electroencephalogram.

In the three remaining cases the fever was part of a severe clinical picture, completed by visceral pains and signs of irritation of the autonomic nervous system. This clinical picture appeared in paroxysmal attacks which were separated by symptom-free intervals. A satisfactory explanation of the disease with its periodic exacerbation could not be found. No efficient causal therapy could therefore be applied. Two of the three patients died presenting very severe abdominal symptoms. In one patient, the finding shortly before death of a mechanical intestinal obstruction threw no light on the cause. There are no autopsy findings.

A review of the literature shows that the authors' observations present a close analogy to the periodic disease described with Reimann. Up to now all the explanations of the cause are unsatisfactory.

From the Authors' Summary.

Changes in Plasma Protein in Favism as Detected by Electrophoresis. P. Careddu, Minerva pediat. **4**:801 (Sept. 30) 1952.

Electrophoresis confirms the precipitation findings in the differential analysis of the serum proteins in the various phases of an acute hemolytic attack in patients with favism. At first, there is a condition of hyperalbuminemia and hypoglobulinemia; this is followed by an increase of gamma globulins, which is indicative of a regeneration of the red blood cells. A deviation from this picture may indicate abnormal regeneration.

Higgins, Boston.

Concentration of Antibiotics in the Sera of the Blood and of Bullae. L. Bessone and G. Bellieni, Minerva pediat. **4**:1059 (Dec. 31) 1952.

Twenty-nine children were used in a study to find out whether antibiotics passed readily from the blood serum into the fluid present in blisters produced by a vesicant applied to the skin of the thighs. The antibiotics studied were penicillin, streptomycin, chloramphenicol (Chloromycetin), and oxytetracycline (Terramycin). Comparative assays of the sera were made one to eight hours after the medication was given. The assays were done by diluting the sera and then testing their bactericidal titer. Each of the antibiotics was found to have passed readily into the bullae, the concentration of antibiotic in the bulla and in the blood being essentially the same. These findings afford a scientific explanation of the brilliant therapeutic results in cases of pemphigus.

Higgins, Boston.

Tumors Seen in Children During the Past Fifteen Years in the Pediatric Clinic of Naples. G. Murano, Pediatria **60**:113 (March-April) 1952.

Of the 8,000 children seen in the hospital during the past 15 years, there have been 36 with tumors. Sixteen, of 44%, had kidney tumors; 12 were in children between 8 months and 3 years of age. In the eight cases in which histological examination was possible, seven tumors were embryonal adenomyosarcomata, and one was a rhabdomyosarcoma. Of the 10 intracranial tumors, 5 were tuberculomata, 1 was a gumma, 3 were neuropliomata, and 1 was a cystic mycosis of the pituitary gland, with diabetes insipidus and Simmonds' cachexia. There were seven cases of lymphosarcoma, 2 cases of Hutchinson's sympathetoblastoma, and one case of osteosarcoma.

Higgins, Boston.

A Radiographic Method of Measuring the Panniculae Adiposus of the Anterior Abdominal Wall. Manuel Suarez, Rev. españ. pediat. **8**:675 (Sept.-Oct.) 1952.

Suarez describes a technique for measuring the subcutaneous fat of abdomen by x-ray. Cuts of various types are included. This type of study is of more value in nutritional research than clinically, the author says.

Allen, Santa Fe, N. M.

TUMORS IN CHILDHOOD. MAXIMILIANO SALAS, Bol. méd. Hosp. inf. **10**:114 (April) 1953.

The author stresses some of the most frequent consequences derived from the unnecessary handling of neoplasms and insists on the necessity of a wider and more intelligent cooperation between the clinician and the pathologist in order to achieve a better knowledge of neoplastic processes.

In the trauma caused by unnecessary manipulation, the author points out the possibility of loosening neoplastic cells, of causing a local congestive process, of bringing about necrosis by hemorrhage and concomitant ulceration, or of throwing into the blood stream unusual amounts of secretion products in certain types of neoplasm.

He insists on the necessity of a biopsy as a means to prove and determine the type of neoplasm before establishing any kind of treatment, this without forgetting the dangers of biopsy, such as hemorrhage, infection, and dissemination of neoplastic cells. Nevertheless, the procedure is justified, since it is the only way to identify the nature of the tumor, although the correct interpretation of the biopsy depends on the experience of the pathologist, on the selection of the tissue to be examined, and on the clinical information given.

When mentioning radiation, the author considers the principal anatomic responses of cells to x-rays of the electromagnetic spectrum, the relation existing between radiumsensitivity and cellular differentiation, the destructive effect of x-rays on the cells, and the relation between the reproductive acitivity of the cells and the destructive effects of the irradiation. The conclusion drawn from all this is that there is a real difficulty in the histologic interpretation of irradiated neoplastic tissues; therefore, there must be a close cooperation of the clinican and the radiologist with the pathologist if a positive benefit is to be obtained from the study of neoplasms. When mentioning the specific effects of hemotherapeutic agents on the malignant neoplasms, the author considers the necessity of always taking into account those modifications caused by said agents on the tissues when morphologic interpretations of the lesions are made.

FROM THE AUTHOR'S SUMMARY.

News and Comment

ANNOUNCEMENTS

National Foundation for Infantile Paralysis.—A nationwide study to determine the effectiveness of a polio vaccine in preventing paralytic polio will get under way in one or more southern states during the week of Feb. 8, 1954. Prior to this date, Dr. Jonas E. Salk, Research Professor of Bacteriology at the University of Pittsburgh—the research grantee of the National Foundation, who developed the vaccine—will have resumed his immunization studies in Allegheny County, Pennsylvania, with vaccination of 5,000 to 10,000 additional children. This was announced today by Basil O'Connor, president of the National Foundation for Infantile Paralysis.

In a gradually expanding program, more than 200 counties throughout the United States will be involved before the mass vaccination project ends by June 1, 1954. During that period, 500,000 to 1,000,000 school children of the second grade will have taken part in one of the largest studies of its kind ever undertaken. Participation will be on a voluntary basis with the consent of the child's parents or legal guardians. Final results of the evaluation study are not expected until some time in 1955.

In general, school children of the second grade will be vaccinated during a nonepidemic period and then observed during a subsequent polio epidemic to evaluate the protective effects of the vaccine. The basic factor for determining the protective effects of the vaccine will be a comparison of the incidence of paralytic polio in the vaccinated group with that of children in the first and third school grades.

In announcing the vaccine study, Mr. O'Connor emphasized that the uniqueness of the project was not merely its size. "For the past 16 years," he said, "millions of Americans have actively supported scientists in the laboratory, helping them create this new potential weapon against polio. And now they will continue that cooperation and help prove whether we have really reached our goal—the conquest of polio."

The vaccine evaluation project will be under the over-all supervision of Dr. Hart E. Van Riper, medical director of the National Foundation. Actively cooperating in the study are medical and scientific societies, state and county public health officers, and school authorities. The Association of State and Territorial Health Officers has appointed a committee consisting of Dr. R. H. Hutcheson (Tennessee), Dr. John D. Porterfield (Ohio), and Dr. Herman E. Hilleboe (New York), to advise with the National Foundation in planning the project.

In the counties selected for the study, local physicians will administer the vaccine under the supervision of the county health officer. Members of the National Foundation's 3,100 chapters will participate as volunteers in providing the many nonprofessional services required in the mass vaccinations. Other community and civic groups also will play a leading role in easing the task of vaccinating a half million or more children in hundreds of communities throughout the nation.

The 200 or more counties to be included in the vaccine validity study are being selected mainly on the basis of a previous polio history that will provide a statistically significant result. The criteria are as follows: (1) high polio incidence for the past five years; (2) high epidemic rate in past five years during June through September; (3) high attack rate in specific age group; (4) adequate health and education facilities; (5) socioeconomic factors, geographic location, etc., to provide a significant cross-section study.

The vaccine to be used in the study is composed of killed virus of all three polio virus types which have been grown in test-tube cultures of monkey kidney tissues. The virus is killed by exposure to formalin and is prepared in a watery solution. This is called the aqueous

type. Each child will receive three injections (1 cc. each) of vaccine, the first two doses at weekly intervals and the third, or "booster shot," four weeks later. The injections will be given in the arm.

In preliminary studies already reported by Dr. Salk, nearly 700 children and adults have been vaccinated without any ill effects or pain. Blood studies on groups of these volunteers have shown that vaccination stimulates the person to develop significant amounts of polio antibodies within a few weeks. From clinical and laboratory studies it has been shown that these antibodies are an index of a person's ability to resist the paralytic disease.

The object of the vaccine study is to determine whether the amount of antibodies resulting from vaccination will prevent paralytic polio in a child who is exposed to the disease under natural conditions. An integral part of the vaccine study is to determine how long such immunity lasts.

The vaccine to be used in the nationwide program will come from several sources. Some of it will be produced by Dr. Salk in his University of Pittsburgh laboratories. The major portion, however, will be produced by pharmaceutical manufacturers according to Dr. Salk's formula. At a recent meeting with pharmaceutical manufacturers they pledged the National Foundation their support and cooperation in setting up production facilities for the manufacture of the vaccine.

Before being administered to any children, each batch of the vaccine will undergo triple safety tests. One of these tests will be conducted by the pharmaceutical manufacturer producing the vaccine; a similar test will also be done by Dr. Salk, and a third test will be made by the Biological Standards Division of the National Institutes of Health, a branch of the U. S. Public Health Service which licenses and controls the manufacture of all biological preparations.

Western Society for Pediatric Research.—The Western Society for Pediatric Research was organized with the following officers: President, John Anderson; Vice-President, Robert Aldrich; Secretary-Treasurer, Forrest Adams; Members of the Council, Vincent Kelley, Frederic Moll, C. Henry Kempe, John McCreary, Robert Chinnock, and Edward Shaw. The first meeting was held on Oct. 29, 1953, at Lane Hall, Stanford University School of Medicine, San Francisco.

PERSONAL

Appointment for Dr. Julius B. Richmond.—Dr. Julius B. Richmond has been made Chairman of the Department of Pediatrics, State University of New York at Syracuse, College of Medicine, Syracuse, N. Y.

Appointment for Dr. H. N. Sanford.—Dr. H. N. Sanford has been made Professor and Head of the Department of Pediatrics at the University of Illinois College of Medicine, Chicago.

Books

Pediatrie. Two Volumes. By Robert Debre and Marcel Lelong. Price, 14,500 francs. Pp. 1,260, with 388 illustrations, and 1,154, with 221 illustrations. Ernest Flammarion, 22 rue de Vaugirad, Paris, 6e, 1952.

This book is a classic. In two loose-leaf volumes it contains more than 2,200 pages of accurate description of children's diseases. A vast amount of material is presented to the practitioner and a wealth of data to the scientific worker. With all recent advances in pediatrics incorporated, the work is up-to-date in every chapter and reflects the rapid development of pediatrics in recent years. The international literature, especially the American, has been thoroughly considered; it is a document of increasing and fruitful international scientific collaboration. The diction is masterful, a splendid example of the old French tradition of cultivated writing. In accord with the development of many special fields in pediatrics, the authors have enlisted the services of 38 collaborators. Experience has shown that textbooks with a large number of individual authors frequently lack uniformity, some chapters being out of proportion. To prevent this, the text of each co-author was "reviewed and adapted." As a result, the presentation is well balanced, in fact, one gets the impression that a single author was responsible for the writing of the entire work.

The textbook is divided into 15 sections. Section 1 deals with congenital diseases, influence of physical factors, allergy, pathology of the newborn, prematurity, toxic conditions, avitaminoses, malnutrition, and intoxications. The role of electrolytes in toxic conditions of infants is fully discussed. A list of solutions for rehydration includes Hartman's, Butler's, and Darrow's solutions. The chapter on rickets brings an adequate discussion of hypervitaminosis D. A valuable chapter on intoxications (34 pages) deals with poisonings from drugs and other chemical products. Section 2 is devoted to gastrointestinal diseases, Section 3 to diseases of the liver and pancreas, and Section 4 to cardiovascular diseases. This section includes a very informative chapter on the development of the heart and an exhaustive discussion of congenital malformations of the heart (45 pages). Section 5 takes up the respiratory diseases. A large number of excellent x-ray pictures is incorporated. Next comes a section on kidney diseases. The final section of Volume 1 brings a clear treatise of skin disorders. An interesting feature of this section is the classification of common skin diseases on the basis of age (early infancy, childhood, puberty). Nobody can deny that age is a determining factor in the appearance of skin disorders in childhood.

Volume 2 contains sections on locomotor disturbances, malignant tumors, tuberculosis, syphilis, disturbances of metabolism, endocrine disorders, diseases of the nervous system, and blood diseases. The chapter on tuberculosis is a masterpiece of clinical presentation. It includes detailed information on BCG—history, tests, technique, control of results, complications, and indications. The diseases of the nervous system are thoroughly covered. An introductory chapter on "generalities" is very informative. The limited space prohibits a more detailed account of the other sections, but all of them give the latest information in their respective fields.

To the reviewer, it is regrettable that two highly important phases of pediatrics are not incorporated in Debre and Lelong's work: infant feeding and acute contagious diseases. The authors mention in their preface that contagious diseases are discussed in another volume of the collection. The reason for omitting nutritional requirements and feeding of normal infants may be that a number of textbooks in this special field are available, however, a textbook on pediatrics without infant feeding and contagious diseases is not complete. These defects do not diminish the great value of the chapters discussed.

Paper, print, and most of the illustrations are excellent. There is an alphabetic index of 21 pages.

The monumental work of Debre and Lelong is a major contribution to pediatrics.

Kitchen Strategy: By Leona M. Bayer, M.D., and Edith Green. Price, $3.75. Pp. 112, with no illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1952.

This loose-leaf booklet was written to convert modern knowledge of nutrition into good eating habits for the family. Its aim is to aid in preparation of meals that will always provide adequate rather than ideal food requirements. In view of the fact that a mother must not only be a dietician but also an economist, hostess, juggler, and chef, the book is designed as a primer of kitchen tactics, giving such information as will enable a mother to conserve her energy and increase her effectiveness. There are sections on leftovers, box lunches, substitutes for restricted foods, coordinating child and family menus, marketing, and diets for various needs such as acne, obesity, diarrhea, constipation, and anemia. There are basic lists of essential food, Calory charts, and even various ways of saving time in meal preparation.

The book is cleverly organized and briefly but well written. It should be a welcome addition to the kitchen library in any house.

Diagnostic Manual in Speech Correction: A Professional Training Workbook. By Wendell Johnson, Frederic L. Darley, and D. C. Spriestersbach. Price, $2.50. Pp. 221. Harper & Brothers, 49 E. 33rd St., New York 16, 1952.

This manual is intended for the use of persons trained in the field of speech pathology. As such, it gives a tremendous amount of information relative to the scope of the professional area of speech disorders. The authors have attempted to provide material which is noncontroversial insofar as this can be done when some of these disorders are still not clearly understood. It forms a much-needed amassment of materials which the trained person may use as much or as little as he likes. In any event, it is an excellent guide to the proper examination of speech disorders.

Rorschach Standardization Study: Development of the Basic Rorschach Score Supplementary Monograph. By Charlotte Buhler, Ph.D., D. Welty Lefever, Ph.D., Frances E. Kallstedt, M.A., and Horace M. Peak. Price, $2.50. Pp. 70, with 19 figures and 14 tables. Rorschach Standardization Studies, 1127 N. Sweetzer Ave., Los Angeles 46, 1952.

The Basic Rorschach Score was developed by Buhler as a means for facilitating standardization of the Rorschach test on different population groups. In 1945 she gave the Rorschach test to persons in 15 different diagnostic groups. The resulting test protocols were scored according to the usual Rorschach scoring determinants. Each determinant was then assigned a "weight." The mean of all these weights in a given protocol was given the name of Basic Rorschach Score. Buhler's principle finding was that the Basic Rorschach Score for each of the 15 groups represented in the study seemed to fall on a continuum, the highest positive Basic Rorschach Score being achieved by the normal group, the highest negative Basic Rorschach Score being achieved by the schizophrenic group. The Basic Rorschach Score, therefore, seemed a usable device for making diagnostic discrimination and also for accumulating standardization data.

In the present monograph, four studies are reported, of which three attempt to test further the validity and reliability of the method described above, and the fourth (Peak) studies the incidence of the popular response (responses presumed to occur in one out of every three subjects) on the Rorschach test in Buhler's 15 diagnostic categories.

In general, the findings of the first three studies (by Lefever, Kallstedt, and Buhler respectively) indicate the Basic Rorschach Score degree of reliability (for diagnostic groups, not necessarily for individual persons) and that it is probably a valid enough measure to be useful in standardization projects.

The reducibility of a given group of homogeneous persons to a numerical score may be of value in large-scale screening of people where such a procedure has meaning. Buhler's technique also opens up avenues for further research among students of the Rorschach, students of psychopathology, and all investigators who do not deal essentially with the individual

clinical evaluation. Buhler and her colleagues correctly point out the danger in applying the Basic Rorschach Score to the individual case. This kind of mechnical misuse of her technique could be analogous to a thermometer reading by the pediatrician which was used to decide the nature, cause, and treatment of a child's disorder.

The Psychoanalytic Study of the Child. Volume VII. Price $7.50. Pp. 447. Edited by R. S. Eissler, A. Freud, H. Hartmann, and E. Kris. International Universities Press, Inc., 227 W. 13th St., New York 11, 1952.

More than half of the 21 articles in this interesting volume are of great value to the practising pediatrician. Especially noteworthy are the following:

Anna Freud—"The Role of Bodily Illness in the Mental Life of Children."

Else Pappenheim and Mary Sweeney—"Separation Anxiety in Mother and Child." This report deals with the treatment of a 4-year-old child who was not ready for nursery school placement.

Lucie Jessner, Gaston E. Blom, and Samuel Waldfogel—"Emotional Implications of Tonsillectomy and Adenoidectomy on Children." This article recounts the observations on 143 children. It is interesting that in 80% the focus of anxiety in children below 4 years of age is the hospital itself, while the operation, injection, and anesthesia are the main problem in only 20% of cases. Interesting also is the observation that for some children under 4 years of age the effect of an overnight stay in the hospital preceding the operation was dubious, while the preoperative hospitalization had a reassuring effect on 50% of the children between 4 and 8 years of age and on 25% of those above 9.

John Bowlby, James Robertson, and Dina Rosenbluth—"A Two-Year-Old Goes to the Hospital." This paper accompanies a film "A Two-Year-Old Goes to the Hospital" made by the Tavistock Clinic in London. Common responses of a child separated at this age for a period of a week from the mother may be as follows: (1) intense clinging to the mother, which can continue for weeks, months, or years; (2) a rejection of the mother as a love object. Such a reaction is only temporary. The documentary film was shown recently by Mr. Robertson in many children's institutions of this country and will be available for purchase and demonstration.

Another group of articles which important pediatric implications covers the following areas: (1) case histories of children treated psychoanalytically—"Masturbation" (M. Bonaparte), "Critical Neurosis in a 2½ Year Old" (S. Fraiberg), "Oedipal Problem" (M. Meiss); (2) psychologic mechanisms—"Sublimation in a Group of 4 Year Old Boys" (M. Harries), "Clinical Notes on Aggression" (D. Beres), "Moral Masochism" (M. Breuman), "Artistic and Infantile Psychoses" (M. Mahler); (3) psychologic-educational problems—"A Survey of Learning Difficulties in Children" (G. Pearson), "Notes on Child Rearing in America" (C. Olden), "Early Child Development in Relation to Degree of Flexibility of Maternal Attitude" (E. Jackson, E. Klatskin, and L. Wilken).

An interesting paper by E. Lindemann and L. G. Dawes deals with the use of psychoanalytic constructs in preventive psychiatry.

Other articles, though important for the progress of psychoanalyietic theory in the field of personality development, are somewhat difficult for the uninitiated to read. I quote as examples the papers by H. Hartmann, W. Hoffer, Anna Freud, and others, discussing "The Mutual Influences in the Development of Ego and Id."

Regarding the volume as a whole, one is impressed by the many interesting subjects discussed lucidly, which must attract and satisfy pediatric readers with varying experience in the field of psychoanalysis.

The New Way to Better Hearing; Through Hearing Reeducation. By Victor L. Browd, M.D. Price, $3. Pp. 226, with 8 pages of color plates and numerous charts and graphs. Crown Publishers, Inc., 419 Fourth Ave., New York, 1951.

The jacket of this book offers "the proved method of overcoming hearing disabilities and learning to hear well with or without professional instruction or hearing aids." Such a statement is bound to arouse suspicion in the scientific mind of an otologist or an audiologist and

could conceivably detract from further perusal of a work which actually has several good features to recommend it.

The chief merit of the book is to be found in the detailed analysis of what the person with poor hearing should know about his own hearing problem. In addition, there are many valuable hints about how a handicapped person can derive the maximum auditory comprehension from the daily situations of life, such as personal communication, group activities, and radio, moving picture, and telephone listening.

Compared with these concrete and factual recommendations the basic principles of hearing reeducation, as outlined by the author, seem to merge into more tenuous concepts. These deal with hidden hearing power, conditioning to situations, and the statement that familiarity lessens difficulty. Numerous exercises in hearing training are outlined under the heading of "Material for practice sessions." After 69 of these, the patient is allegedly rehabilitated and should have no further difficulty which cannot be resolved by a brief refresher course. Whether the benefits claimed for these exercises can be scientifically corroborated remains an unanswered question.

Chemie und Klinik der Bilirubinreduktionsprodukte Urobilin und Sterkoblin. By Rudolph Horst, M.D. Price, 8 Deutsche marks. Pp. 61, with 13 illustrations. Georg Thieme, Thomaskirchof 21, (10 b) Leipzig C1, agent for U. S. A., Grune & Stratton, Inc., 381 4th Ave., New York 16, 1952.

This little book is devoted exclusively to the metabolism and disorders of the metabolism of bile pigment. The little slip of the pen describing the epithelium of the liver as ectodermal had better be corrected. The author leans heavily on Baumgärtel, who sees the difference of the bilirubin giving the direct van den Bergh reaction from that giving the indirect one in that the direct-reacting bilirubin is held in colloidal solution by means of bile acids. In all probability the work of Najjar was not published in time to receive consideration. In a chapter on formal genesis of icterus, a relationship of glycogen metabolism to icterus is stressed, accepting the differentiation of Baumgärtel into glycogenotoxic and plasmatoxic agents. The glycogenotoxic agents produce a high degree of impoverishment of the cell in glycogen with fat infiltration and necrosis. The sugar formed facilitates the entrance of bilirubin into the blood, with usually severe icterus. The plasmatoxic agents which may be produced by various infections produce hardly any impoverishment in glycogen and a light icterus, while a destructive dissociation of the liver cells can occur with little fat infiltration. The reduction products of bilirubin; mesobilirubin, the precursor of urobilinogen; urobilinogen, and stercobilinogen are treated in several subchapters. In accordance with Baumgärtel, the transformation of bilirubin in urobilinogen is done by cell ferments, while stercobilinogen owes its origin to intestinal bacteria. Urobilinogen is formed where bile stagnates. It is found in the bile of the gall bladder and the ducts leading to the duodenum, not in the hepatic ducts. Urobilinogen is resorbed from the duodenum; its precursor, mesobilirubin, from the jejunum. These bilirubinoids could be demonstrated in portal blood (page 23), while (page 35) mesobilirubin is exclusively found in the gall bladder. The resorbed urobilinogen is destroyed in the liver under normal conditions and is not excreted in the urine. In the feces it appears rarely under abnormal conditions. Neither urobilinogen nor stercobilinogen is subject to enterohepatic circulation. Stercobilinogen being resorbed from the lower large intestine into the hemorrhoidal veins enters the urine normally and is increased whenever a pleiochromic bile enters the intestine, as, for instance, in familial hemolytic jaundice. Both urobilinogen and stercobilinogen give the Ehrlich aldehyde reaction, and their oxidation products, urobilin and stercobilin, the Schlesinger reaction. Various methods to differentiate these substances are cited, but for practical purposes the mesobiliviolin reaction is recommended, which by the author's modification of chloroform extraction can be carried out on as little as 10 ml. of infant's urine. It is based on the fact that mesobiliviolin is obtained by treating the residue of the chloroform extract with boiling concentrated hydrochloric acid containing some ferric chloride. It is soluble in chloroform from which the blue color can be extracted by dilute sodium hydroxide. This reaction differentiates urobilin from stercobilin, which treated in the same manner yields a product which does not enter the dilute sodium hydroxide and imparts to the chloroform a yellow color. This reaction can also be carried out with properly prepared fecal extracts. Urobilinogen may also undergo a change to propentdyopent, so that the urine gives the red pentdyopent reaction which

is discussed. Stercobilinogen does not give the reaction. The clinical material consists of the examination of 141 persons. Icterus catarrhalis as a hepatosis is treated separately from epidemic hepatitis. In cases of physiologic icterus neonatorum the stools contained urobilin, but the urine contained neither bilirubin nor urobilin. Stercobilin was absent, since no bacteria had invaded the intestines. In cases of icterus gravis neonatorum (erythroblastosis) a trace of bilirubin and urobilin was found in the urine which was free from stercobilin. The results of the examination of urine and stool in the various disorders are tabulated with reference to the presence of urobilin and stercobilin at various stages of the disorder. Whenever urobilinogen is found in the urine, it means an interference with the natural bilirubin excretion. This happens with injuries to the liver parenchyma and in mechanical bile stasis. A short graph illustrates the differences in bilirubin in blood and urine and of urobilin and stercobilin of urine and feces compared with the norm in cases of parenchymatous and obstructive jaundice and hemolytic icterus. Two cases of liver cirrhosis in infants are reported in greater detail. In one of these, where the cirrhosis was probably due to an atresia of the bile ducts, in spite of the icterus, bilirubin was absent from the urine and urobilinogen was present only in traces, but an unusual strong pentdyopent reaction was obtained. Stercobilinoids were much diminished in the urine and the stool. It is assumed that the degradation of bilirubin proceeded rapidly to propentdyopent or to preformed pentdyopent.

The little book contains much of interest.

Standard Values in Blood: Being the First Fascicle of a Handbook of Biological Data. Edited by Errett C. Albritton, A.B., M.D. Price $4.50. Pp. 199. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1952.

This report presents in tabular form standard values in blood for man and other vertebrates. These are arranged under the following sections: physical properties; coagulation phenomena; blood groups; erythrocytes and hemoglobin; leukocytes; blood and bone marrow cells; water; carbohydrates, lipids, and proteins; amino acids and nonprotein nitrogen compounds; phosphorus and sulfur compounds; vitamins, hormones, and enzymes; electrolytes, minerals, gases, and acid-base values; effects of radiation and storage; effective blood levels of therapeutic agents.

These very comprehensive data are supplemented with essential information on nomenclature, units, methods and conditions of measurements, investigated strains, and theories. An extensive bibliography using a special reference system is appended which also contains the name of the contributor for each value.

The Battle for Mental Health. By James Clark Moloney, M.D. Price, $3.50. Pp. 105. Philosophical Library, Inc., 15 E. 40th St., New York 16, 1952.

Dr. Moloney is a psychiatrist and psychoanalyst in adult practice who early became interested in the origins of mental health, or ill health, in infancy. He was one of the group of psychiatrists, pediatricians, obstetricians, and other professionals who founded the Cornelian Corner in Detroit in order to study and foster such trends as self-regulation of feeding, breast feeding, and the rooming-in of newborn infants with their mothers, which seemed to offer hope of bettering child-parent relationships. He observed the emotional stability of the Okinawans under severe stress in war, which he attributed largely to the warm permissiveness with which they were cared for in infancy.

In this book Dr. Moloney makes a vigorous plea for more warmth and permissiveness in infant care in America as one antidote for our considerable mental and social ill health. He lays the blame on such factors as rigidity in infant feeding, overemphasis on early toilet training, the submissiveness of one type of mother, the dominatingness of another, and what he believes was the subservience of American pediatrics to the arbitrary and authoritative spirit of German pediatrics around the turn of the century. He also indicates such American ideals as excessive competitiveness, perfectionism, and machine-like efficiency for human beings.

He counterattacks, with gusto, those who have resisted the movement toward naturalness in infant care, particularly obstetricians "with misanthropic attitudes toward the newborn," nursery nurses who are cold and stubborn, rigid pediatricians, and parents who are excessively conforming or narcissistic.

For remedies Dr. Moloney proposes a lying-in nursing home completely removed from the general hospital not only in location, but also in atmosphere and spirit. He also suggests that all the experts cease criticizing American mothers, and that their husbands give them more emotional support.

Those who already share Dr. Moloney's philosophy will find his supporting material interesting and will enjoy the indignation with which he writes. They might complain of the narrowness of his argument, since he omits from his line-up in the battle for mental health such institutions as the school, the college, the church, the social agency, and the settlement house and considers no other period of childhood than infancy.

Those who by temperament reject anything new or anything permissive until it is thoroughly proved will not be convinced by this book; they will, more likely, be enraged.

The Course of Tuberculosis in the Body. By Reiner W. Müller. Price, 24 Deutsche marks. Pp. 170, with 55 partly multicolored illustrations. Georg Thieme, Diemershaldenstrasse 47, (14a) Stuttgart-O; agent for U. S. A. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1952.

The author's experience derives from his connection with a big sanatorium for children in Württemberg, the children's hospitals for tuberculosis of the city of Cologne, and his service during the war in Russia. The object of the genetic analysis in phthisiology is the study of the spatial and temporal sequence of tuberculous changes in man and animal. The differentiation of the type of tubercle bacillus in a given infection, human or bovine or avian, is demanded. The primary infection, the spread of the disease, super infection, and reinfection are detailed, with consideration of the various pathways of spreading and their interrelation. The localizations of the disease in various organ systems and their interrelationships are discussed. The influence of the age of the patient on the disease is portrayed and the influence of the age or, better, duration of the disease on its further course. The regularity in the deposition of lime salts and the diagnosis and differential diagnosis of calcareous depositions are depicted with attention to the differences in various animals. It may be mentioned here that veterinary tuberculosis is given due consideration throughout the monograph. It is of interest to the pediatrician that the author takes sides with those who do not regard erythema nodosum and phlyctenular conjunctivitis as exclusively of tuberculous origin. Neither is he convinced of the tuberculous nature of sarcoidosis, for reasons cited. The author realizes that the advances in therapy already have changed the course of the disease and that further changes are to be expected. The monograph is instructive.

Handbook of Orthopaedic Surgery. Edition 4. By Alfred Rives Shands. Price $8. Pp. 644, with 191 illustrations. C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1952.

In this fourth edition, Dr. Shands has brought the subject matter up to date and has added sections in some 10 subjects on which knowledge and thinking is currently changing. The bibliography, which should be of use to students, has been brought up to date as of March 1, 1952.

The name "Handbook" is still appropriate, for this book is definitely not a reference work. It is very complete as an outline and study book for medical students, but it does not provide exhaustive treatment of any of its subjects. The author is to be commended for his excellent judgment in deleting the obsolete and treating controversial subjects with brevity, while making it clear that all authors are not in agreement. The omission of fresh fractures, which are better handled in a separate volume, and the curtailment of interesting but unnecessary historical background, is commendable in a book of this scope.

The illustrations, mostly clear drawings, have been improved by the addition of x-ray reproductions and some new drawings. In a few cases, the illustrations of deformities would be more realistic if they had been taken from actual patients rather than from poses by a normal model.

This handbook is recommended to the medical student, especially in schools where it is used as the outline for covering the orthopedic courses. The general practitioner will find it a good means of rapidly acquainting himself with the essentials of orthopedic conditions which he meets. A more exhaustive study will require reference books and the use of the bibliography provided by Dr. Shands in this handbook.

Manual of Electrocardiography. By B. F. Smith, M.D. Price $4.50. Pp. 215, with 119 illustrations. Elsevier Press, Inc., 402 Lovett Blvd., Houston, Texas, 1952.

This volume, the result of the author's lectures to his students, is well written and an excellent exposé at the beginner level.

The autopsy reports with the electrocardiograms are valuable. The correlation between the pathological findings and the electrocardiogram could be clarified.

Penicillin Decade, 1941-51: Sensitization and Toxicities. By Lawrence Weld Smith, M.D., and Ann Dolan Walker, R.N. Price $2.50. Pp. 117. Arundel Press, Inc., Box 2606, Washington 13, D. C., 1951.

The authors in this small monograph succeed in a detailed analysis of the different reactions to penicillin. This decade of the use of penicillin is divided into three epochs: first, a period of use of a crude penicillin of low potency which, however, established its relatively low toxicity; second, the "golden era," 1942-1945, when purity improved purity and great clinical effectiveness, while reactions during this period were in part due to menstrua used as oil, wax, etc.; nevertheless, there were reactions to the penicillin as well; third, the period of development of pure crystalline penicillin G, which is being followed by a possible fourth period, dominated by repository dosage with penicillin procaine and more recently others, as L-ephenamine penicillin.

As the authors state, "Antibiotics are only of value for the treatment of infections due to organisms sensitive to the specific antibiotics being employed." The organism must be identified and its sensitivity tested. Unfortunately, the delay of determination may be serious; at least skin sensitivity laboratory tests should be done, especially in patients who have previously received antibiotic therapy.

While penicillin is relatively nontoxic, the ability it has to sensitize may cause serious complications.

This volume should be read by every practicing physician.

Medical Inspection of School Children. By Edgar Henry Wilkins. Price, $4.00. Pp. 224, with 21 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1952.

This is a manual designed for the use of the school health officer in Great Britain. The author is obviously a man of considerable experience in the evaluation of the child as a whole. Chapters are included on the sociological background, clean linens, clothing and vermin, past medical history and accommodation, and other requirements for school medical inspection. In addition, there are chapters covering every phase of physical examination, including growth, posture, gait, dental deformities, and diet.

The book is small, well written, and nicely printed. It would be valuable reading, not only for school health officials, but for any physician dealing with children.

Essentials of Infant Feeding for Physicians. By Herman Frederick Meyer, M.D. Price, $7.75. Pp. 249, with 13 Illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1952.

This monograph was written with the express intention of presenting the principles of infant feeding in the simplest possible terms and a manner which would make those principles easily and practically applicable. As the author remarks, the number of preparations currently available is so great as to make infant feeding seem complex, whereas actually the subject is fundamentally simple.

As the title implies, an attempt is made to present the essentials of infant feeding. In this respect, no deficiencies can be found in the subject matter. No practicing physician would ask for more information than is supplied. After brief chapters on the history of infant feeding and breast feeding, the chemical principles involved are discussed. After each section—fats, proteins, carbohydrates—there is a brief summary of these principles. Mechanisms of infant feeding, mainly prevention of air swallowing, are briefly but adequately considered. There is a short chapter on complemental or mixed feedings, followed by one on formula computation.

A chapter that will be welcome to all who deal in the feeding of infants is the detailed one on proprietary infant foods. Every food available is discussed, and tables are included to give quick available information on the many proprietary foods which few physicians can keep in mind.

There are chapters on solid food supplements, vitamin supplements, feeding problems, and even on self-demand feeding and rooming-in. Much additional miscellaneous information is contained in the appendix.

There have been many books published on the subject of infant feeding, none of which, this reviewer is aware, meets so completely the needs of the practicing physician, whether he be general practitioner or pediatrician. It is an admirable book and is highly recommended.

The Conference on European B. C. G. Programmes. Conducted with the assistance of the Joint Enterprise. Price not given. Pp. 333, with numerous illustrations. The International Tuberculosis Campaign, 25 Svanemøllevej, Copenhagen, Denmark, 1950.

This volume describes in detail the formation of the Joint Enterprise for the purpose of carrying on mass BCG programs on an international scale. The Joint Enterprise was created as the result of action taken by the UNICEF (United Nations International Children's Emergency Fund) at Lake Success on March 12, 1948. It is a cooperative effort between the UNICEF and the Scandinavian Voluntary Organization (Danish Red Cross and its Norwegian and Swedish associates). The World Health Organization is also considered a partner, since it loans technical assistance through its expert committee on tuberculosis. In addition the WHO Research Office, under the direction of Dr. Carroll E. Palmer, of the U. S. Public Health Service, will be furnished with all the statistical information emanating from the BCG program.

Following the section describing the organizational setup are reports on the progress of the BCG campaigns in Greece, Austria, Poland, Yugoslavia, Czechoslovakia, and Finland. As of June 30, 1949, a total of 10,657,891 persons had been tested to tuberculin in the Joint Enterprise project and 5,468,412 vaccinated with BCG.

The third section of the report is devoted to a discussion of tuberculin testing by Dr. H. J. Ustvedt. The advantages and disadvantages of the Moro patch test, the Mantoux, Pirquet, and Trambusti tests are presented in great detail. Which to use in order to be most certain that persons free from tuberculosis are selected for BCG vaccination is gone into with great thoroughness. Then follows an equally fascinating treatment on follow-up testing. Dr. Ustvedt takes great pains to point up all the problems and questions that remain to be answered before knowledge can be final regarding BCG vaccination.

This volume can be highly recommended as very profitable reading to anyone interested in tuberculosis.

The Doctors Jacobi. By Rhoda Truax. Price, $3.50. Pp. 270. Little, Brown & Company, 34 Beacon St., Boston 6, 1952.

In recent years, a number of biographies of leading physicians appeared in English literature. Semmelweis, Ehrlich, and Cajal have been the subject of books. The author of the volume under consideration wrote on Lister. "The Doctors Jacobi" makes a valuable addition to medical biography. Although the author is not a physician, she gives an accurate account of the medical achievements of Dr. Abraham Jacobi, the father of American pediatrics, and of his wife, Mary Putnam Jacobi, who made notable contributions to neurology and to the medical education of women in this country. The author must have done a great deal of research on the subject, as the data is quite accurate. In many cases, she documents her statements in the form of notes at the end of the book.

Miss Truax has given not only the medical aspects of the work of the "Doctors Jacobi," but also the story of the human side of these two physicians, their happiness and their sorrows. Her description of the death of Dr. Jacobi's only son, at the age of seven, is touching. The story of the fire of Jacobi's house, where his autobiography and other personal documents were destroyed, makes fine reading. The same is true of the personal life, joys, and sorrows, of Mary Putnam Jacobi. The author has woven into her book the history of American medicine and Dr. Jacobi's friendship with leading medical men of the country, such as

Osler, Welch, and Garrison. Of great interest is the reference to Dr. Jacobi's boyhood friend, Carl Schurz, who, like Dr. Jacobi, was a German immigrant and was imprisoned in Germany because of revolutionary activities, and who, like Dr. Jacobi, has played a great role in American life, although in a different field of endeavor.

Pediatricians, particularly, should find a great deal of interest in the book "The Doctors Jacobi."

Nutritional Studies in Adolescent Girls and Their Relation to Tuberculosis. By Joseph A. Johnston. Price, $7.50. Pp. 330, with 194 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1953.

The author of this book is the Pediatrician-in-Chief at the Henry Ford Hospital and the Edsel B. Ford Institute for Medical Research, in Detroit. For many years, his work on the nutritional and metabolic aspects of adolescence has been outstanding.

The book is valuable because it brings together in one volume a number of the author's excellent studies covering a 20-year period. It presents balance data of the food requirement and the various factors which influence its utilization in the adolescent. Major portions of the text are devoted to records of observations on individual children. There is an extensive discussion of the factors which affect retention of nitrogen and calcium in this important period of growth. The interrelationship between the endocrine glands, rest and activity, and of focal infection as well as nutrition are clearly presented.

The author emphasizes the importance of the interplay between growth and nutritional requirements in relationship to disease and its manifestations.

As a result of these numerous studies and the correlation of available published data, the author attempts to show that the abrupt increase in the adult form of tuberculosis in adolescence is related to a failure to meet the nutritional requirements for growth peculiar to this period. By the presentation of a number of case records, he shows that a favorable course of the disease process, once it has developed, may be conditioned to a large extent by the degree to which we succeed in promoting a normal nutritional state and replenishing previously acquired deficits.

The appendix includes diets. There is a short but pertinent bibliography. Unfortunately, there is no index. However, the table of contents is sufficiently detailed to make up somewhat for this omission.

This text should be familiar to all pediatricians, nutritionists, and students of growth. The book is "a must" for those who deal with tuberculosis in this age group.

Improving The Child's Speech. By V. A. Anderson. Price, $5.50. Pp. 333. Oxford University Press, 114 5th Ave., New York 11, 1953.

This book gives a summary of some of the most frequently met speech and hearing disorders in the public schools intended to provide classroom teachers with elementary information concerning these defects. Such information is badly needed by regular school teachers because of the heavy incidence of these handicaps in the school population. Because of the limitations of the audience in regard to basic information in sociology, psychology, and medicine, the book contains very little analysis of these speech disorders, except in a more or less superficial way. It presents selective methods of correction, which are in the nature of selected techniques that may or may not produce the results desired. The medical problems presented by many of these case are not covered by this text, and the many neurological disorders which are involved in the production of speech and hearing handicaps are not discussed except in very brief form. The reader who wishes a general overview of the problem could probably get a great deal of help from this book. Any person who wishes information as to cause and specific methods of rehabilitation should look at other texts which are intended for a different reading audience. Subjects covered are the nature and development of speech, recognition of speech disabilities, special problems of children with these handicaps, and general advice concerning integrating such training with the school curriculum.

Directory of Pediatric Societies *

INTERNATIONAL

International Union for Child Welfare

President: Mrs. Gordon Morier.
Secretary: Dr. Georges Thélin, 16 rue du Mont Blanc, Geneva, Switzerland.

IV° Congreso Pan-Americano de Pediatria

President: Dr. Leonel Gonzaga.
General Secretary: Dr. Adamastor Barbosa.
Secretariat: Rua Dias da Rocha 25, ap. 602, Copacabana, Rio de Janeiro.
Place: Rio de Janeiro. Time: 1954.

IV° Congreso Sudamericano de Pediatria, Confederacion Sudamericano de Sociedades de Pediatria

President: Prof. Carlos Prado.
Vice-President: Dr. Marcelo Garcia.
General Secretary: Dr. Alvaro Aguiar.
Place: Avenida mem de Sá 197, Rio de Janeiro. Time: 1954.

FOREIGN

Academy of Medicine, Toronto, Section of Paediatrics

Chairman: Dr. Bernard Laski, Toronto.
Secretary: Dr. Crawford Anglin, Toronto.
Place: Academy of Medicine.

Asociación de Pediatras Españoles

President: Dr. Rafael Ramos Fernández.
Vice-Presidents: Dr. Juan Bosch Marín and Dr. Ciriaco Laguna.
Secretary-General: Dr. Luis Navas Migueloa.
Treasurer: Dr. Angel Ballabriga.

Australian Paediatric Association

President: Dr. P. A. Earnshaw, Ballow Chambers, Wickham Terrace, Brisbane. B17.
Honorary Secretary-Treasurer: Dr. D. C. Jackson, 217 Wickham Terrace, Brisbane. B17.
Place: Canberra. Time: Yearly in April.

British Paediatric Association

President: Prof. J. Smellie.
Secretary: Prof. Alan Moncrieff, Institute of Child Health, The Hospital for Sick Children, Great Ormond St., W.C. 1, London.
Place: Windermere. Time: Between Easter and Whitsunday.

Canadian Paediatric Society

President: Dr. D. B. Leitch, 210 McLeod Bldg., Edmonton, Al.
Secretary: Dr. R. L. Denton, 1615 Cedar Ave., Montreal 25.

Danish Pediatric Society

President: Dr. A. Rothe-Meyer, Children's Hospital, Fûglebakken, Copenhagen F.
Secretary: Dr. E. Winge Flensborg, Children's Hospital, Martinsvej, Copenhagen V.
Place: Different hospitals. Time: First Wednesday of every month.

Departamento de Pediatria da Associação Paulista de Medicina

President: Dr. J. Leme da Fonseca.
Secretary: Dr. Joaquim da Costa Marques.

Departamento de Puericultura e Pediatria da Associação Medica do Parana

President: Dr. Haroldo Beltrao.
Secretary: Dr. Mucio de Ábreu Neto.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Deutsche Gesellschaft für Kinderheilkunde

President: Prof. Dr. med. B. de Rudder, Frankfort/Main, Universitäts-Kinderklinik.
Secretary: Prof. Dr. med. Jochims, Städtische Kinderklinik, Lübeck.

Hellenic Society of Pediatrics

President: Dr. Spiros Charocopos, Alexandras Ave. 1, Athens.
Secretary: Dr. Nikas Zervos.
Place: Amphitheater, Medical School of the University of Athens. Time: Once a month.

Nederlandse Vereeniging voor Kindergeneeskunde

President: Dr. G. J. Huët, 39 Brediusweg, Bussum.
Secretary: Dr. H. Reerink, % Sophia Kinderziekenhuis, 160 Gordelweg, Rotterdam.
Place: Children's Hospital or Pediatric Clinic. Time: Three times a year.

Norsk Pediatrisk Selskap

President: Dr. Arne Njå, Sporveisgt. 29, Oslo.
Secretary: Dr. Helge Granrud, Abbedikollen 38, Bestun, Oslo.
Place: Barneklinikken, Rikshospitalet, Oslo. Time: Friday, six times a year.

Pediatric Society of New Zealand

President: Dr. Edward J. Cronin, Power Board Bldg., Queen St., Auckland.
Secretary: Dr. J. D. Matthews, 104 Symonds St., Auckland.

Philippine Pediatric Society

President: Dr. Renato Ma. Guerrero.
Secretary: Dr. Felix A. Estrada.
Place: Santo Tomas University Hospital and other pediatric centers. Time: Every two months.

Royal Society of Medicine, Section of Paediatrics

President: Dr. C. T. Potter.
Honorary Secretaries: Dr. A. P. Norman and Ursula James, M.R.C.P.
Sectional Editor: Dr. D. MacCarthy.
Place: 1 Wimpole St., London W.1 (Correspondence for these honorary officers should be sent to this address). Time: Fourth Friday of each month, 4: 15 p.m.

Schweizerische Gesellschaft für Pädiatrie

President: P. D. Dr. F. Bamatter, St.-Léger 8, Genève.
Secretary: Dr. M. Nicod, Place Bel-Air 2, Lausanne.

Sociedad Argentinade Pediatrie

President: Dr. Rudolfo Kreutzer.
Secretary: Dr. Luis M. Cucullu.
Place: Sante Fè 1178, Buenos Aires.

Sociedad Boliviano de Pediatria

President: Dr. Carlos Daza Meruvia.
Secretary: Dr. Cecilio Abela Deheza.
Place: S. C. I. S. P. Bldg. Time: Every Thursday.

Sociedade Brasiliera de Pediatria

For officers, see IV° Congreso Sudamericano de Pediatria, above.

Sociedad Chihuahuense de Pediatría

President: Dr. Alfonso Ruiz Escalona.
Secretary: Dr. Armando G. Herrera.
Place: Hospital Infantil del Estado, Chihuahua, Chih., Mexico.

Sociedad Chilena de Pediatria

President: Prof. Dr. Raúl Gantes A.
Secretary-General: Dr. Edmundo Cardemil.
Place: Medical Society, Merced 565, Santiago.

Sociedad Colombiana de Pediatria

President: Dr. Jorge Camacho Gamba.
Vice-President: Dr. Antonio Alvarez Riaño.
Secretary: Dr. Hernando Vanegas A.
Treasurer: Dr. Efrain Perry Villate.

Sociedad Cubana De Pediatria

President: Prof. Agustin Castellanos y Gonzales.
Secretary: Dr. Carlos Hernandez Miyares, Apartado 2430, Habana.
Place: Catedra de Clinica Infantil Hospital Nuestra Senora de las Mercedes and Hospital Municipal de Infancia, Habana. Time: 9 p.m., last Wednesday of every month.

Sociedad de Pediatría de Concepción (Chile)

President: Dr. Próspero Arriagada.
Secretary: Dr. Guillermo Repetto D.
Place: Hospital Clinico Reginal Concepción. Time: 7:30 p.m., second Tuesday of every month, April to December, inclusive.

Sociedad de Pediatria del Litord

President: Dr. Raul Bruera.
Vice-President: Dr. Isidoro Slullitel.
Secretary-General: Dr. Elsa Galimany.

Sociedad de Pediatria y Puericultura del Atlantico

President: Dr. Atilio Marino.
Vice-President: Dr. Helena Uribe de Jimenez.
Secretary-Treasurer: Dr. Hernando Borrios Angulo.

Sociedade de Pediatria da Bahia

President: Dr. Elysio Pereira de Atbayde.
Secretary: Dr. Eliezer Audiface.

Sociedade de Pediatria de Bernambuco

President: Dr. Antonio Aureliano.
Secretary: Dr. Claudia Figueiredo.

Sociedade de Pediatria do Rio Grande do Sul

President: Dr. Francisco Talaia de Moura.
Secretary: Dr. Enio Pilla.

Sociedade de Puericultura e Pediatria do Maranbão

President: Dr. Odorico Amaral de Mattos.
Secretary: Dr. Joao Mohana.

Sociedade Mineira de Pediatria

President: Dr. Agostinho Fernandes.
Secretary: Dr. Celio Marques Scotti.

Sociedade Portugesa de Pediatria

President: Prof. Dr. Lucio de Almeida.
Vice-President: Dr. Cordeiro Ferreira.
Secretary-General: Prof. Dr. Carlos Salazar de Sousa.
Treasurer: Dr. Mario Cordeiro.
Place: Clinica pediatrica do Hospital D. Estefania, Lisbon. Time: Monthly.

Sociedad Uruguaya de Pediatria

President: Dr. Euclides Peluffo.
Secretary: Dr. Ruben Gorlero Bacigalupi.
Place: Av. Agraciada 1464, piso 13, Montevideo.

SOCIEDAD VENEZOLANA DE PUERICULTURA Y PEDIATRÍA

President: Dr. Guillermo Rangel.
Secretary: Dr. Guillermo Tovar E.,, P.O. Box 5, Caracas.
Place: Hospital Municipal de Niños, Caracas. **Time:** Once a month.

SOCIEDAD YUCATECA DE PEDIATRIA

President: Dr. Humberto Perez Concha.
Secretary: Dr. Prudencio Ruiz Salazar.
Treasurer: Dr. Jorge Antiveros Andrade.

LA SOCIÉTÉ DE PÉDIATRIE DE MONTRÉAL

President: Dr. Henri Charbonneau, 3095 Sherbrooke, Montréal.
Secretary: Dr. Edmond Baril, 301 Carré St-Louis, Apt. 1, Montréal.
Time: Second Thursday of each month.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Prof. Jean Cathala, 45 rue Scheffer, Paris XVI.
Secretary: Prof. Maurice Lamy, 94 rue de Varenne, Paris VII.
Place: Hôpital des Enfants Malades, 149 rue de Sèvres, Paris XV. **Time:** 4 p.m., third Tuesday of each month.

SOCIÉTÉ DE PÉDIATRIE DE QUÉBEC

President: Dr. Roland Thibaudeau, 203 des Erables, Québec.
Secretary: Dr. Euclide Dechêne, 312 du Roi, Québec.
Place: Quebec. **Time:** May and November.

SOUTH AFRICAN PAEDIATRIC ASSOCIATION

Chairman: Dr. Seymour Heymann, 804 Medical Centre, Jeppe St. Johannesburg.
Honorary Secretary-Treasurer: Dr. I. Kessel, Transvaal Memorial Hospital for Children, Milner Park, Johannesburg.

SWEDISH ASSOCIATION OF PEDIATRICS

President: Prof. Sture Siwe, Lund.
Secretary: Dr. Karl-Axel Melin, Färjestadsvägen 16, Bromma.

SWEDISH MEDICAL SOCIETY, SECTION FOR PEDIATRICS AND SCHOOL HYGIENE

President: Docent Urban Hjarne, Stockholm.
Secretary: Dr. M. d'Avignon, Pediatric Clinic, Karolinska Sjukhuset, Stockholm 60.
Place: Swedish Medical Society, Klara Östra Kyrkogata 10, Stockholm, and Pediatric Clinic, Uppsala. **Time:** Second Friday of each month, except summer.

UNITED STATES

National

AMERICAN MEDICAL ASSOCIATION, SECTION ON PEDIATRICS

Chairman: Dr. Eugene T. McEnery, 4458 W. Madison St., Chicago 24.
Secretary: Dr. Wyman C. C. Cole, 3001 W. Grand Blvd., Detroit.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. Roger L. J. Kennedy, 102 2d Ave., S. W., Rochester, Minn.
Vice-President: Dr. Crawford Bost, 115 Terrace Dr., San Francisco 27.
Executive Secretary: Dr. E. H. Christopherson, 636 Church St., Evanston, Ill.
Treasurer: Dr. Edgar E. Martmer.

SOCIETY FOR PEDIATRIC RESEARCH

President: Dr. Robert Ward.
Vice-President: Dr. Russell Blattner, Houston, Texas.
Secretary-Treasurer: Dr. Sydney S. Gellis, Boston.

THE AMERICAN PEDIATRIC SOCIETY

President: Dr. Rustin McIntosh.
Vice-President: Dr. Wilburt C. Davison.
Secretary-Treasurer: Dr. Aims C. McGuinness.

Sectional

Intermountain Pediatric Society

President: Dr. John T. Mason.
Secretary: Dr. Stanley R. Child.
Place: Ambassador Athletic Club, Salt Lake City. Time: 7 p.m.

New England Pediatric Society

President: Dr. Albert W. Fellows.
Secretary: Dr. Harry Shwachman, 300 Longwood Ave., Boston.
Place: Boston, except Spring meeting. Time: Wednesdays, four times a year.

North Pacific Pediatric Society

President: Dr. Carl Ashley, 2455 N. W. Marshall St., Portland, Ore.
Secretary-Treasurer: Dr. S. Gorham Babson, 1107 S. W. 13th St., Portland, Ore

Northwestern Pediatric Society

President: Dr. F. G. Hedenstrom, 420 Lowry Medical Arts Bldg., St. Paul.
Secretary: Dr. Charles L. Steinberg, 1118 Lowry Medical Arts Bldg., St. Paul.
Place: Minneapolis; St. Paul; Duluth, Minn., and Rochester, Minn. Time: January, April, July, and October.

Rocky Mountain Pediatric Society

President: Dr. John Lichty, Pediatric Department, Colorado General Hospital, 4200 E. 9th Ave., Denver.
Secretary: Dr. Edwin T. Williams, 1850 Gilpin St., Denver 6.
Place: Announced by mail. Time: Monthly dinner meetings, October to June.

Southwestern Pediatric Society

President: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills, Calif.
Secretary: Dr. Carl A. Erickson, 960 E. Green St., Pasadena, Calif.
Place: Jonathan Club of Los Angeles. Time: 6:30 p.m., second Wednesday in January, March, May, September, and November.

Western Society for Pediatric Research

President: Dr. John Anderson.
Vice-President: Dr. Robert Aldrich.
Secretary-Treasurer: Dr. Forrest Adams.

State

Alabama Pediatric Society

President: Dr. David B. Monsky, 748 Adams St., Montgomery 5.
Secretary: Dr. Alice H. Pye, 305 Church St., Montgomery.
Place: Montgomery.

Arizona Pediatric Society

President: Dr. Henry E. Running.
Secretary: Dr. William F. Schaffman.
Time: On Call.

California Medical Association, Section on Pediatrics

Chairman: Dr. Joseph W. St. Geme, 511 S. Bonnie Brae, Los Angeles 5.
Secretary: Dr. Alvin H. Jacobs, 2201 Webster St., San Francisco 15.
Assistant Secretary: Dr. Clement J. Molony, 416 N. Bedford Dr., Beverly Hills.

Florida State Pediatric Society

President: Dr. Charlotte C. Maguire, 1413 Kuhl Ave., Maguire Bldg., Orlando.
Secretary-Treasurer: Dr. Wesley S. Nock, 273 Alhambra Circle, Coral Gables.

Georgia Pediatric Society

President: Dr. John A. Simpson, Athens.
Secretary: Dr. J. Harry Lange, 490 Peachtree St., Atlanta.
Place: Savannah. Time: At time of annual state meeting.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Joseph Hetzel, 51 Central Ave., Waterbury.
Secretary: Dr. James H. Root Jr., 103 N. Main St., Waterbury.
Time: With the Connecticut State Medical Society in the spring, with the Connecticut State Clinical Congress in the fall; winter meeting usually in December.

Illinois State Medical Society, Section on Pediatrics

Chairman: Howard R. Miller, 418 Jefferson Bldg., Peoria 2.
Secretary: Harry H. Boyle, 2376 E. 71st St., Chicago 49.

Indiana State Pediatric Society

President: Dr. Harold D. Lynch, Evansville.
Secretary: Dr. L. T. Meiks, Indianapolis.

Iowa Pediatric Society

President: Dr. Charles J. Baker, 403 Carver Bldg., Fort Dodge.
Vice-President: Dr. P. E. Gibson, Davenport Pediatric Group, Davenport.
Secretary-Treasurer: Dr. Ralph E. Dyson, 3200 University Ave., Des Moines.
Place: Spring meeting in conjunction with the Iowa State Medical Society meeting. Fall meeting at University Hospital, Iowa City.

Massachusetts Hospital Association

President: Dr. Philip D. Bonnet, Administrator, Massachusetts Memorial Hospitals, Boston.
Executive Secretary: Henry G. Brickman, 14 Somerset St., Boston 8.

Michigan State Medical Society, Pediatric Section

President: Dr. Horace L. French, 301 Seymour Ave., Lansing.
Secretary: Dr. Park S. Bradshaw, 1014 Jefferson St., Muskegon.

Montana Pediatric Society

President: Dr. Orville M. Moore, 555 Fuller Ave., Helena.
Secretary: Dr. Joseph W. Brinkley, 410 Central Ave., Great Falls.
Place: Variable. Time: Spring and fall.

Nebraska Pediatric Society

President: Dr. Michael Crofoot.
Vice-President: Dr. Donald C. Nilsson.
Secretary-Treasurer: Dr. A. J. Lombardo, 401 Aquila Ct., Omaha.
Place: Children's Memorial Hospital. Time: 6 p.m., dinner, third Thursday of each month.

New Hampshire Pediatric Society

President: Dr. Douglass W. Walker, Laconia.
Secretary: Dr. Wm. D. Penhale, Concord.
Place: Alternately Concord, Hanover, Laconia, and Manchester. Time: Four times a year.

New Mexico Pediatric Society

President: Dr. M. K. Wylder, Suite 14, Medical Arts Sq., Albuquerque.
Secretary: Dr. Ly Werner, Suite 25, Medical Arts Sq., Albuquerque.
Place: Variable. Time: Every two or three months.

North Carolina Pediatric Society

President: Dr. Eugene Keiter, Kinston Clinic, Kinston.
Vice-President: Dr. Donald Robinson, 308 W. Davis St., Burlington.
Secretary-Treasurer: Dr. Robert Pope, Wilson Clinic, Wilson.
Place: Roaring Gap and Chapel Hill. Time: Sept. 12 and Oct. 30, respectively.

South Carolina Pediatric Society

President: Dr. Walter Moore Hart, 117 W. Cheves St., Florence.
Secretary: Dr. John R. Paul Jr., 82 Rutledge Ave., Charleston.
Place: Columbia, S. C. Time: Second Monday and Tuesday in September.

Tennessee Pediatric Society

President: Dr. O. L. Von Canon, 846 McCallie Ave., Chattanooga.
Secretary: Dr. Oliver W. Hill, 4807 Newcom Ave., Knoxville.

Texas Pediatric Society

President: Dr. Robert L. Moore, Dallas.
President-Elect: Dr. M. C. Carlisle, Waco.
Secretary: Dr. James N. Walker, Fort Worth.
Treasurer: Dr. Jack M. Woodall, Big Springs.

Virginia Pediatric Society

President: Dr. Edwin L. Kendig Jr.
Secretary: Dr. T. J. Humphries.

Wisconsin State Medical Society, Section of Pediatrics

Chairman: Dr. E. H. Pawsat, Fond du Lac.
Vice-Chairman: Dr. Samuel E. Kohn, Milwaukee.
Secretary: Dr. J. R. Schroder, Janesville.
Delegate: Dr. K. J. Winters, Wauwatosa.
Alternate Delegate: Dr. F. Mellencamp, Milwaukee.

Local

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. C. T. J. Dodge, 15457 Euclid Ave., Cleveland 12.
Secretary: Dr. Beulah Wells, 10515 Carnegie Ave., Cleveland 6.
Place: Allen Memorial Medical Library. Time: 8:15 p.m., October, December, February, and March.

Baltimore City Medical Society, Pediatric Section

President: Dr. Wilson Grubb, 4 E. 33d St., Baltimore 18.
Secretary: Dr. Gibson J. Wells, 3101 St. Paul St., Baltimore 18.
Place: 1211 Cathedral St., Baltimore 1. Time: 8:30 p.m., second Tuesday in November, January, and March.

Bronx Pediatric Society

President: Dr. M. H. Grand, 1749 Grand Concourse, New York.
Secretary: Dr. Charles T. Fried, 1220 Grand Concourse, New York.
Place: Morrisania City Hospital. Time: 8:30 p.m., second Wednesday of each month, except June, July, August, and September.

Brooklyn Academy of Pediatrics

President: Dr. Stanley S. Lamm, 20 Plaza St., Brooklyn.
Secretary: Dr. Henry Cohen, 1761 E. 19th St., Brooklyn.
Place: Granada Hotel, Brooklyn. Time: 8:45 p.m., fourth Wednesday of February, March, April, October, and November.

Buffalo Pediatric Society

President: Dr. Douglas P. Arnold.
Secretary: Dr. Richard A. Downey.
Place: Children's Hospital, 219 Bryant St. **Time**: 8:30 p.m., first Monday of each month, September to June.

Central New York Pediatric Club

President: Dr. Frank Williams, 58 Swan St. S., Albany.
Secretary: Dr. Fred W. Bush, 325 Park Ave., Rochester.
Place: Various cities in New York State; fall meeting jointly with Upper New York State Academy of Pediatrics. **Time**: Third Tuesday of April and October.

Central Ohio Pediatric Society

President: Dr. O. W. Hosterman, 350 East Broad St., Columbus 15.
Secretary-Treasurer: Dr. John Riepenhoff, 2998 Sullivant Ave., Columbus.
Place: As announced. **Time**: Third Wednesday of September, November, January, March, and May.

Chicago Pediatric Society

President: Dr. Alfred S. Traisman.
Vice-President: Dr. Eugene T. McEnery.
Secretary: Dr. Matthew M. Steiner, 6354 N. Broadway.
Treasurer: Dr. Maxwell P. Borovsky.
Place: Children's Memorial Hospital, 710 Fullerton Ave. **Time**: Third Tuesday of each month, October to May, inclusive.

Cincinnati Pediatric Society

President: Dr. Joseph E. Ghory, 2546 Auborn Ave.
Secretary: Dr. Clare R. Rittershoffer, 301 Erkenbrecker.
Place: Children's Hospital, Elland Ave., Cincinnati. **Time**: On call.

Cleveland Academy of Medicine, Pediatric Section

Chairman: Dr. Robert Hauver, 10515 Carnegie Ave.
Secretary: Dr. William Herman, 10300 Carnegie Ave.

Corpus Christi Pediatric Society

President: Dr. Edwin A. Danford.
Secretary: Dr. Jack F. McKemie.
Place: As announced. **Time**: 7:30 p.m., fourth Wednesday of January, March, May, July, September, and November.

Dade County Pediatric Society

President: Dr. Lynn Whelchel, DuPont Bldg., Miami, Fla.
Secretary: Dr. Howard Engle, 311 Lincoln Rd., Miami Beach, Fla.

Dallas Pediatric Society

President: Dr. Floyd A. Norman, 4325 Cole Ave., Dallas.
Secretary-Treasurer: Dr. Charles L. Smith, 4207 Lemmon Ave., Dallas.
Place: Children's Hospital of Texas. **Time**: 6:30 p.m., first Thursday in each month.

Detroit Pediatric Society

President: Dr. Ruben Meyer, 938 Maccabees Bldg., Detroit.
Secretary: Dr. W. W. Zuelzer, Children's Hospital of Michigan, Detroit.
Place: Variable. **Time**: Five times a year.

Honolulu County Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Corresponding Secretary and Treasurer: Dr. John H. Peyton, Kauikeolani Children's Hospital, Honolulu.

Honolulu Pediatric Society

President: Dr. Teruo Yoshina, 1221 Victoria St., Honolulu.
Recording Secretary: Dr. M. H. Hasegawa.
Corresponding Secretary: Dr. John Peyton, Kauikeolani Children's Hospital, Honolulu.
Place: At doctors' homes in rotation. Time: 6 p.m., third Thursday of each month.

Houston Pediatric Society

President: Dr. Emanuel B. Brandes, 3101 Fannin St., Houston.
Secretary: Dr. Robert D. Lomas, 5101 Fannin St., Houston.
Place: River Oaks Country Club. Time: 7:30 p.m., fourth Tuesday of each month.

Kansas City Southwest Pediatric Society

President: Dr. Frank Hogue, Plaza Medical Bldg., Kansas City, Mo.
Secretary: Dr. S. J. Kranson, Truman Rd. and Ash, Independence, Mo.
Place: University Club, Kansas City, Mo. Time: On call.

Long Beach Pediatric Society

President: Dr. Kenneth Berkaw, 211 Cherry Ave., Long Beach, Calif.
Secretary: Dr. C. W. Latshaw, 3821 Atlantic Ave., Long Beach, Calif.
Place: Victor Hugo Restaurant, 730 E. Broadway. Time: First Tuesday night of every month.

Los Angeles County Medical Association, Pediatric Section

President: Dr. Sidney Rosin, 6230 Wilshire Blvd., Los Angeles.
Secretary: Dr. Arthur J. Holmes, 65 N. Madison Ave., Pasadena 1.
Place: Los Angeles County Medical Association Bldg., 1025 Wilshire Blvd., Los Angeles. Time: Second Thursday of alternate months beginning in September.

Louisville Pediatric Society

President: Dr. Lee Palmer.
Secretary: Dr. Martin Z. Kaplan.
Place: Variable. Time: Five times a year.

Medical Society of the County of Kings and Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Morton Seldowitz, 1245 Union St., Brooklyn.
Secretary: Dr. Bernard Greenberg, 755 Ocean Ave., Brooklyn 26.
Place: Kings County Medical Society Auditorium, 1313 Bedford Ave., Brooklyn. Time: 8:45 p.m., fourth Monday of January, March, April, October, and November.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. George Maksim, 1418 Good Hope Rd. S. E., Washington 20.
Secretary: Dr. John E. Cassidy, 5402 Connecticut Ave. N.W., Washington.
Place: Medical Society Bldg., 1718 M St. N.W., Washington. Time: Spring and fall.

Memphis Pediatric Society

President: Dr. James G. Hughes, 20 S. Dunlap, Memphis.
Secretary: Dr. George S. Lovejoy, 609 N. Highland, Memphis.
Place: Rotated among four hospitals. Time: 7:30 p.m., second Tuesday of each month.

Milwaukee Pediatric Society

President: Dr. Franklin Mellencamp, 1513 E. Capitol Dr., Milwaukee.
Secretary: Dr. Karl Beck, 1411 Wauwatosa Ave., Milwaukee.
Place: Milwaukee University Club. Time: Second Wednesday of February, April, June, and October.

Mobile Pediatric Society

President: Dr. Daniel Sullivan, 1420 Government St., Mobile, Ala.

Secretary: Dr. Robert O. Harris III, 1514 Government St., Mobile, Ala.

Place: Admiral Semmes Hotel. Time: 7:30 p.m., fourth Thursday of September, November, January, March, and May.

Nashville Pediatric Society

President: Dr. Thomas Weaver, Bennie Dillon Bldg., Nashville, Tenn.

Secretary: Dr. Ethel Walker, 3415 West End Ave., Nashville, Tenn.

Place: Homes of members. Time: Third Thursday of every month.

Nassau Pediatric Society

President: Dr. Martin Glynn, 41 Holyoke Rd., Rockville Centre, N. Y.

Secretary: Dr. David J. Posner, 98 Wolcott Rd., Levittown, N. Y.

Place: Garden City Hotel, 7 & Park Ave., Garden City, L. I., N. Y. Time: 7:30 p.m., second Monday of October, December, February, April, and June.

New York Academy of Medicine, Section on Pediatrics

Chairman: Dr. Jerome L. Kohn, 50 W. 96th St., New York 25.

Secretary: Dr. Lawrence B. Slobody, 1 E. 105th St., New York 12.

Place: New York Academy of Medicine, 2 E. 103d St. Time: 8:30 p.m., second Thursday of each month, November to May, inclusive.

Northern California Pediatric Society

President: Dr. John A. Anderson, Stanford Hospital, 2351 Clay St., San Francisco 15.

Secretary: Dr. Edward Campion, 1018 E St., San Rafael.

Place: San Francisco. Time: Quarterly, as speakers are available.

Oklahoma City Pediatric Society

President: Dr. C. M. Bielstein, 301 N.W. 12th St., Oklahoma City.

Secretary: Dr. Louis Frank, 1200 N. Walker St., Oklahoma City.

Place: Oklahoma Club. Time: On call.

Philadelphia Pediatric Society

President: Dr. John B. Bartram, 2600 Lawrence St., Philadelphia 33.

Secretary: Dr. F. H. Harvie, 133 S. 36th St., Philadelphia 4.

Place: College of Physicians, 19 S. 22d St. Time: 8:30 p.m., second Tuesday from October to May, inclusive.

Pittsburgh Pediatric Society

President: Dr. Christian John Stoecklein, 519 Medical Arts Bldg., Pittsburgh 13.

Secretary-Treasurer: Dr. James W. Stirling, 694 Lincoln Ave., Pittsburgh 2.

Place: Western State Psychiatric Institute and Clinic. Time: Second Friday in October, December, February, and April.

Queens Pediatric Society

President: Dr. Samuel Dillon, 119-20 Rockaway Beach Blvd., Rockaway Park, N. Y.

Secretary: Dr. Moe Goldstein, 111-32 76th Ave., Forest Hills, N. Y.

Place: Seminole Club, 112th St. and 70th Rd., Forest Hills, N. Y. Time: Second Tuesday in October, December, February, and April.

Richmond Pediatric Society

President: Dr. Hobart T. Dougan, 1832 Monument Ave., Richmond 20, Va.

Secretary: Dr. Robert A. Hoffman, 1001 W. Franklin St., Richmond 20, Va.

Place: Medical College Hospital. Time: 8 p.m., third Thursday of each month, except June, July, and August.

San Antonio Pediatric Society

President: Dr. Edith M. Bonnet, 228 Medical Arts Bldg., San Antonio 5.
Secretary: Dr. Virginia S. Stovall, 1116 S. Texas Bldg., San Antonio 5.
Time: 7:30 p.m., supper, first Tuesday of each month.

Seattle Pediatric Society

President: Dr. O. William Anderson.
President-Elect: Dr. Robert Tidwell.
Secretary-Treasurer: Dr. James L. Tucker.
Place: College Club. Time: 6:30 p.m., fourth Friday of each month from September to June.

St. Louis Pediatric Society

President: Dr. H. E. Wachter, 8505 Delmar.
Vice-President: Dr. Raymond J. LaDriere, 35 No. Central, Clayton 5, Mo.
Secretary-Treasurer: Dr. Leo F. Gowen, 337 W. Lockwood, Webster Groves 19, Mo.
Place: Medart's Restaurant. Time: 7 p.m., second Thursday of each month from September to May, inclusive.

Santa Barbara Pediatric Society

President: Dr. Clarence B. Hills, 22B. W. Micheltorena St., Santa Barbara, Calif.
Secretary: Dr. Louise P. Brown, 3017 Foothill Rd., Santa Barbara, Calif.
Place: Varies. Time: Two to three times yearly, on call.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Fred J. Chapin, 101 John St., Bay City.
Secretary-Treasurer: Dr. George H. Lowrey, University Hospital, Ann Arbor.
Place: University Hospital, Ann Arbor.

Waco Pediatric Society

President: Dr. J. M. Garrett, 1408 Austin Ave., Waco, Texas.
Secretary: Dr. C. G. Shellenberger, 1318 Austin Ave., Waco, Texas.
Place: 1917 Austin Ave., Waco, Texas. Time: Third Tuesday of each month.

Westchester County Medical Society, Pediatric Section

President: Dr. Herbert Kahan, 650 Main St., New Rochelle, N. Y.
Secretary: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: The Roger Smith Hotel, White Plains, N. Y. Time: Four dinner meetings a year, November, February, April, and June.

Western Michigan Pediatric Society

President: Dr. A. Morgan Hill, 310 Fulton, E., Grand Rapids.
Secretary: Dr. John C. Montgomery, 1810 Wealthy St., S.E., Grand Rapids.
Place: Two meetings in Grand Rapids, two elsewhere. Time: First Wednesday of October, November, April, and May.

Wichita Pediatric Society

President: Dr. Frank L. Menehan.
Secretary: Dr. R. C. Knappenberger.
Place: Homes of members. Time: 8:30 p.m., first Thursday, October through May.

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1. Tisdall, F. F., and Jolliffe, N., in Clinical Nutrition, New York, P. B. Hoeber, 1950, c. 23, p. 590. 2. Sealock, R. R., and Goodland, R. L.: Science *114*: 645 (Dec. 14) 1951.

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REFERENCES

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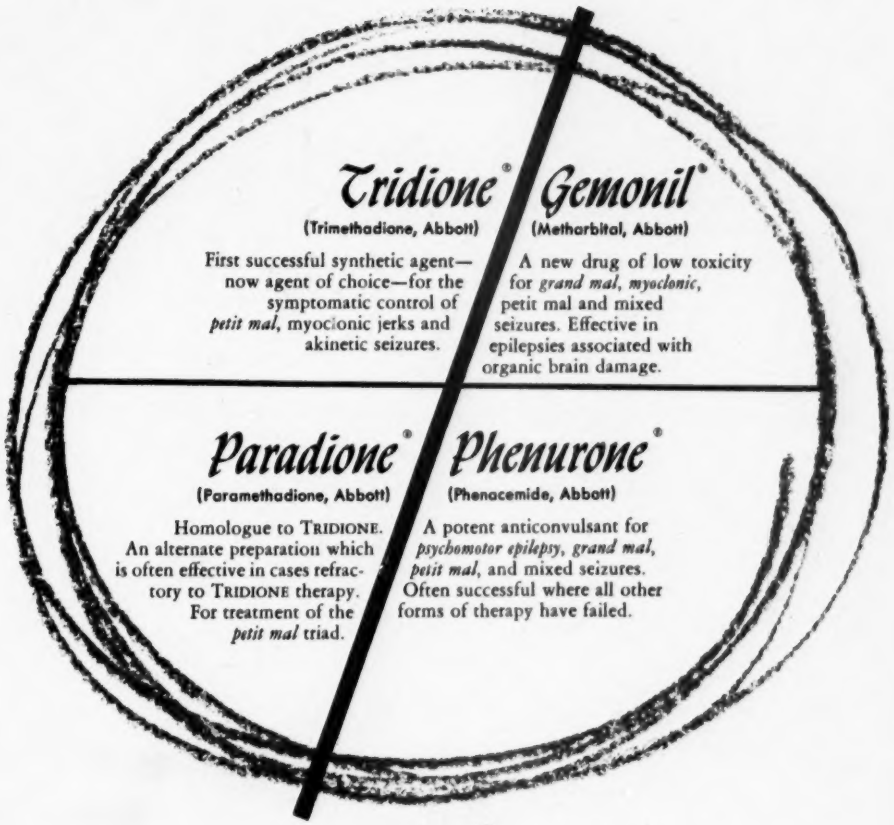
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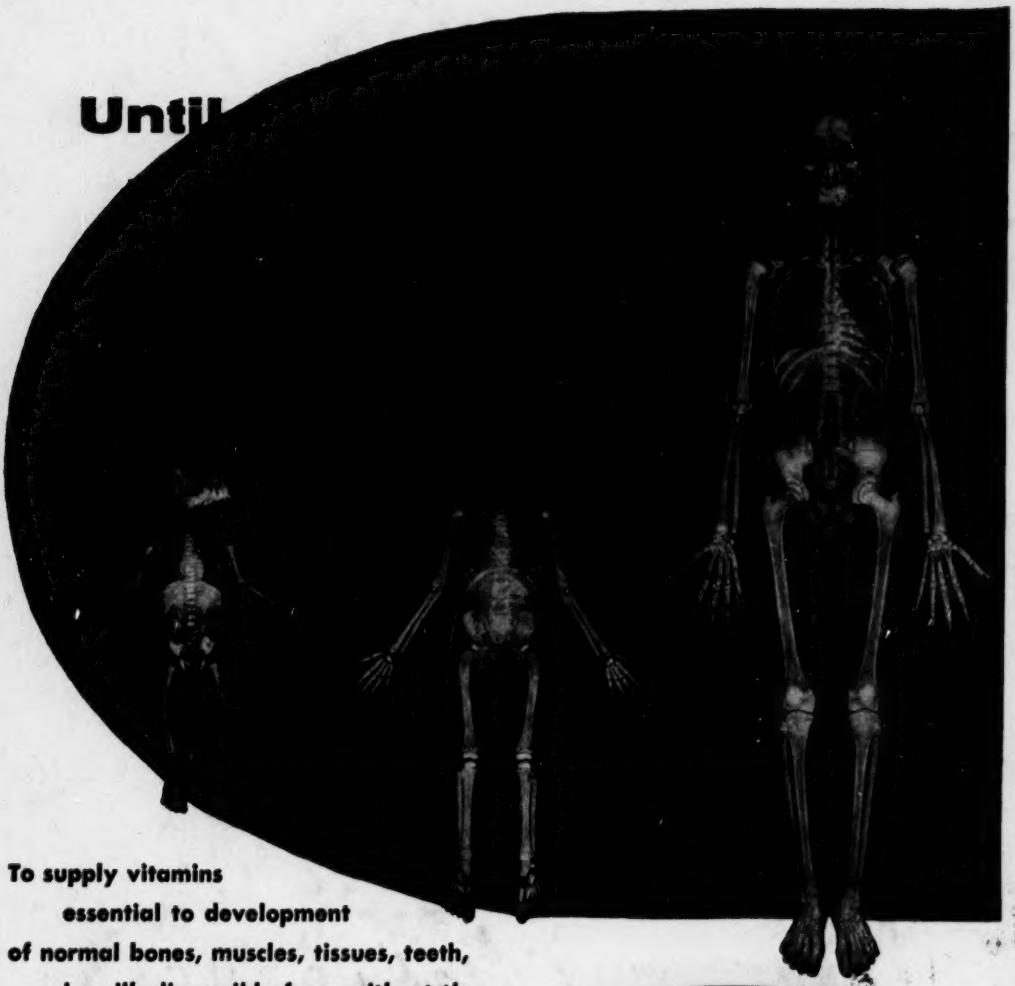

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